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EDITORIAL

"ACTUAL OR BOOKKEEPING MATERNAL DEATHS?"

FOR many years we have had drilled into us the fact that the United States has, and has had, a high maternal mortality. In fact, things are so deplorable that we are far down in the list of nations, it being much safer for a woman to have her child in any of several countries than our own. Because of this there has been much talk, and the naming of countless committees to study conditions and report—a few years back the New York Academy of Medicine brought in a report that was a sensation and led to acrimonious debate.

It is conceded that things might be a great deal better. We know that obstetrical teaching in this country is nothing to look to with great pride. In some institutions of learning, the undergraduate in medicine sees less than three babies born before he is weaned and given a degree. We've heard the argument that the midwives are at fault, that every woman should go for her delivery to a well equipped maternity hospital, that every woman should have her baby at home unless there are apparent physical abnormalities present, that physicians should take postgraduate courses in midwifery and get up to date, that they should develop an obstetrical conscience—the propositions and suggestions are endless.

Not so long ago, we spent part of an afternoon attending and listening to a maternal mortality survey committee at its work. In this city two men from each hospital attend the meetings. All deaths of pregnant women, as recorded by the Board of Health, are looked into, and a digest of all findings are read at the meeting. After a reading of the record had ended, the question was asked: "Was death due to the fault of the patient, the physician in charge, or to both?"

In arriving at the answer, one heard a splendid scientific discussion that dealt with many obstetrical problems. At the

end of the discussion a vote was taken and the blame, if any, was placed at the proper door.

During this discussion certain important facts were revealed—at least, they seemed important to us. It seemed that after a certificate of death was filed, a clerk in the health office made a record of the maternal death, and the cause, for future filing and statistical reports. But, upon study, in a true sense, some of the fatalities should not have been recorded as maternal deaths. In other words, a woman was insane before she became pregnant. She was insane during her pregnancy. Two weeks post partum she died, the result of her type of insanity. This case was recorded as a maternal death. Another example: a woman five months pregnant contracted influenza and pneumonia and died. The records had it listed as a maternal death. A woman six months pregnant was operated on for acute appendicitis. She went into labor, delivered a premature infant, and died of peritonitis. This was classified as a maternal death. We learned that in the majority of cases, the fault lies with the physician who makes out the death certificate. In his hurry, or by taking the easiest way out, he does not fill out the certificate properly and does not write in the facts. The Health Department can do no other than file the death as a maternal one from the findings of the physician's signed certificate. That afternoon four of twenty deaths put in the maternity mortality column did not belong there—an error of twenty per cent.

All this brings us to a query; how accurate are the records from which the maternal mortality statistics in the United States are computed? How careful are physicians and house-doctors of institutions in filling out these certificates? How many physicians are aware that from the records they file our mortality figures are arrived at, and what percentage of American physicians know how to fill out a death certificate properly?

Is our actual maternal mortality truly as high as reported? From what we have observed and learned, we doubt it.

The care of the prospective mother has a long way to go before it will reach a state bordering on the ideal. Obstetrical teaching in this country must be improved, and the hours allotted to it increased. The teachers themselves must be better prepared and fitted to teach others. The undergraduate and intern must have more clinical work under competent overseers. We would have the fight for better and more competent obstetrical care go on relentlessly.

But meanwhile we would have some organization, private or governmental, study our maternal mortality records, and thereby discover if all maternal deaths truly are maternal deaths, or bookkeeping deaths—women who happened to have been pregnant and died from some cause or causes that had no bearing on this condition.

Perhaps, after all, we are not as badly off in this respect as we have been led to believe.

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ORIGINAL ARTICLES

METYCAINE SPINAL ANESTHESIA*

REPORT OF 1381 CASES

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GENERAL CLINICAL CHARACTERISTICS

IMPROVEMENT in spinal anesthesia will probably come through the use of drugs that have longer action and less toxicity than the drug most commonly used at present, namely procain (trade names, novocain, neocain, spinocain). In our search at the Lahey Clinic for a better drug we have used metycaine with satisfactory results for spinal anesthesia in over 1500 cases, and have found it superior to procain in duration of anesthesia, and clinically no more toxic than that drug. In fact, in the matter of fatalities metycaine has a better record in our hands than does procain, for we have had no deaths attributable to it. Our preference for it as compared to procain is so definite that as we became accustomed to using it we discontinued the use of procain completely.

In other fields, moreover, it appears preferable to commonly used drugs. Thus in local infiltration and regional anesthesia it is effective more promptly and surely than is procain, yet appears to be no more toxic. For topical application in the urethra and in the eye it is probably superior to cocain. In the urethra it is sufficiently effective in 4 per cent solution and less toxic,[10,12] and in the eye in 1 per cent solution in physiologic saline it produces anesthesia without dilating the pupil or desiccating the cornea.[8] It would appear to be a most useful drug for the physician to have in his office because solutions keep without deterioration over a period of months, they are self-sterilizing,[15] and they are effective by topical application as well as by the methods of infiltration and of nerve block.

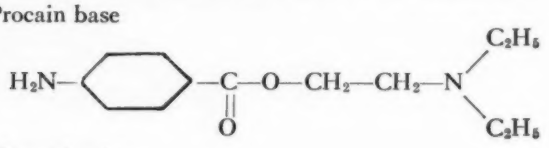
PHYSICAL AND PHARMACOLOGICAL PROPERTIES

Metycaine was synthesized by McElvain[3] of the University of Wisconsin. It has previously been distributed under the names of L.A. 33, neothesin, and pipercaine. It is the hydrochloride of gamma —(2-methyl-piperidino)—propyl benzoate and is chemically related to procain and cocain (Fig. 1). It is a white crystalline powder,[1] easily soluble in water, alcohol and chloroform and does not deteriorate with exposure to air or to sunlight. Aqueous solutions are faintly acid to litmus, remain stable after boiling or sterilizing by autoclave, and are compatible with epinephrin. Pharmacological studies carried out on animals in the Lilly Research Laboratories[2,10] show it to be of approximately the same toxicity as procain when injected subcutaneously, and from two to four times as toxic when injected intravenously. Meeker[4] compared its anesthetic potency with that of procain by raising wheals in the human skin with solutions of each drug. Anesthesia from 1/16 per cent solution of procain lasted five minutes as contrasted with fourteen minutes from the same con-

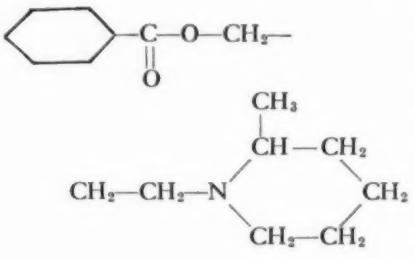
* From the Department of Anesthesia, Lahey Clinic, Boston, Mass.

centration of metycaine. Weaker solutions of procain were ineffectual, whereas metycaine produced anesthesia in dilutions down

Procain base



Metycaine base



Cocain base

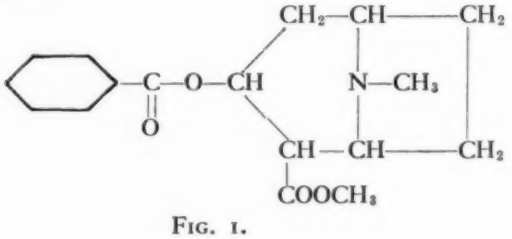


Fig. 1.

to $\frac{1}{128}$ per cent. When combined with small amounts of epinephrin (5 drops of 1:1000 epinephrin in 100 c.c. of solution) procain in ½ per cent solution gave anesthesia for seventy minutes and metycaine for ninety-six minutes.

PREVIOUS REPORTS

Romberger and Ratcliff[9] have reported 2 spinal anesthesias with metycaine, Meeker[5,6] reported 28, and in 1932 I issued a preliminary report[14] covering 43 cases. The Council on Pharmacy and Chemistry of the American Medical Association has approved of the drug for infiltration anesthesia, but has withheld action on it in regard to spinal anesthesia until more information should be available.

Source of Data. The present report covers 1381 administrations, the total number during four years on which sufficient data are available. All anesthesias were administered by members of the Department of Anesthesia of the Lahey Clinic or by graduates in medicine working under their instruction. For each case a detailed record was made of the patient's condition before and during anesthesia and of the technique of administering the anesthetic. From these records were compiled the data which follow.

TECHNIQUE, PATIENTS AND ANESTHETIC RESULTS

Technique and Dosage. Ampoules containing slightly over 2 c.c. of 10 per cent aqueous solution of metycaine were used for most cases. Each patient was placed in the left lateral decubitus, and the skin and interspinous region of the third, or sometimes of the fourth, lumbar interspace were infiltrated with a solution containing ephedrine (usually 50 mg.) and metycaine (1 per cent). Puncture was made with a gold needle of 21 gauge so ground as to have no cutting edge, but sharp at the very point only, like a seamstress' needle. After the stylet had been withdrawn, a 5 c.c. syringe containing the desired amount of 10 per cent solution of metycaine was attached to the needle, spinal fluid was aspirated into the syringe until the desired dilution was obtained, and the mixture immediately injected without barbotage, usually at the rate of 0.5 c.c. per second. The patient was then quickly placed on his back. Like procain, metycaine mixed with spinal fluid forms a solution heavier than spinal fluid alone. This fact is most helpful in obtaining the desired height of anesthesia, because by the effect of gravity the solution may be made to flow cephalad or caudad during the first fifteen minutes after injection.

Details of our technique varied considerably, but in general we used for anesthesia of the perineum from 40 to 80 mg. of metycaine (0.4 to 0.8 c.c. of 10 per cent solution) diluted to 1.5 to 3.0 c.c. with spinal fluid, with the table level. For lower abdominal operations we used from 80 to 140 mg. (0.8 to 1.4 c.c.) diluted to 4 or 5 c.c., with a downward slope of 10 or 15 degrees from sacrum to shoulders, and for upper abdominal operations from 150 to 200 mg. (1.5 to 2.0 c.c.) diluted with spinal fluid to 5 c.c., with a downward slope of

10 to 20 degrees from sacrum to shoulders. Immediately after injection the patient was turned on his back, and the head was flexed forward on the chest so as to place it higher than the shoulders, in order to prevent gravity from pulling the heavy solution cephalad to the medulla. As soon as anesthesia reached the desired height, the table was leveled in order to check the effect of gravity. This usually occurred in three to eight minutes. In each case the age, vigor and weight of the patient, as well as the height and length of anesthesia desired, were considered in determining the amount of metycaine to be used.

After twenty minutes it was considered that the drug was no longer movable by gravity. From this time on, the Trendelenburg position was used if needed to accommodate the surgeon or to combat a fall of blood pressure. If pressure fell below 50 per cent of its preanesthetic level, we ordinarily gave 0.2 c.c. of 1:1000 solution of epinephrin intramuscularly and this was usually followed by a prompt though transient rise in pressure. If the pressure became very low, or marked pallor or any embarrassment in breathing developed, we administered oxygen from a gas machine, using the closed method with absorption of carbon dioxide by soda-lime.[13]

Table I
DOSAGE GROUPS

	Group I 30–95 Mg.	Group II 100–145 Mg.	Group III 150 Mg. Up	Totals
No. cases.....	176	499	706	1381
Per cent of cases.	12.7	36.2	51.1	100.0

In the presentation of data in the sections which follow, all cases are divided into three groups according to whether the amount of metycaine given was small, medium or large (Table 1). Only 10 patients were given very large doses, that is over 200 mg. Most of these were large vigorous patients who were to have gallbladder operations. Three of the 10 did not receive their large amount as a single dose: the first dose of 200 mg. or less failed to give sufficient anesthesia, so a second dose was added, bringing the totals up to 225, 300, and 360 mg. In one of these 3, thoracic breathing, as contrasted to diaphragmatic breathing, was paralyzed during anesthesia, in another it was weak, and in the third it was not affected. Circulatory depression was not severe in any of these 3 patients, the systolic pressures dropping 26, 32, and 28 per cent respectively, from the preanesthetic levels, and in all 3 convalescence was normal. In a fourth case the patient was given 320 mg. at a single dose, probably by mistake, and his shoulders were lowered. Systolic pressure dropped from 124 to 110 only, then rose, without stimulants, to 165, and gradually dropped to 124 again. During the peak of anesthesia one arm was paralyzed, the patient was unable to swallow, and sleep supervened. One hour after injection these symptoms had completely disappeared. Convalescence was normal except for hemorrhage on the seventh day. These 4 cases appear to demonstrate a reasonably wide margin of safety.

Operation. Table II lists the sites of operations. Over 70 per cent were abdominal, with 21 per cent on the upper abdominal group of stomach, biliary tract and spleen. Since high spinal anesthesia entails far more risk than does low anesthesia, this proportion of upper abdominal operations is of importance in interpreting the proportion of cases in which respiration and circulation were depressed, as given in other tables.

Age. Distribution of age is given in Table III. It is of interest to note that there were 29 patients in their eighth decade and 27 between the ages of five and fourteen years. In children the doses of preliminary narcotics and of spinal anesthetic drugs were according to Robson's schedule for procain,* and very satisfactory results were obtained.

* Approximately 1 mg. of morphine per year of age, and 1 mg. of procain per pound of body weight.

TABLE II
OPERATIONS

Site	No.		Per Cent
Thoracoplasty		1	0.1
Laparotomy		981	71.1
Stomach	8		
Gall bladder and bile ducts	280		
Liver	1		
Spleen	1		
Small gut	27		
Large gut (including resection of rectum, excluding appendix)	163		
Appendix	216		
Pelvic organs	237		
Exploration and lysis of adhesions	32		
Sympathectomy	8		
Retroperitoneal tumor	2		
Postcecal abscess	1		
Secondary closure of wound	5		
Kidney and ureter		42	3.0
Herniorrhaphy		64	4.6
Pelvic and perineal (not laparotomy)		271	19.6
Transvaginal (dilatation and curettage, hysterectomy, interposition, etc.)	63		
Transurethral (prostatectomy, cystoscopy, etc.)	99		
External genitalia	37		
Rectum and anus	72		
Lower extremities		20	1.4
Miscellaneous superficial		7	.5
		1386	100.3*

* Over 100 per cent, because some operations involved more than 1 region.

TABLE III
AGE

Age in Years	No.	Per Cent
5– 9	2	0.1
10–14	25	1.8
15–19	49	3.6
20–69	1276	92.4
70–79	29	2.1
	1381	100.0

Risk. In determining the operative risk that each patient presents, every available item that might affect the ultimate outcome of anesthesia and of operation is considered. These include, in addition to other items, the age, vigor, degree of anemia, of dehydration or of starvation, cardiac and respiratory reserve, disease of liver, of kidneys, of metabolism, fever, length of confinement to bed, and prognosis of the surgical disease. Risk 1 means that no condition is known to be present that would affect the outcome unfavorably. Risk 2 means that some factor or factors of moderately unfavorable influence are present, whether they are part of the surgical disease or not. Such factors might be age of sixty years or beyond, a head cold, or moderate anemia. Risk 3 is a danger signal, and means that the patient is in such poor condition that any depressing form of anesthesia or any markedly shock-producing type of operation should be avoided if possible. Risk 4 means that a fatal outcome may be expected. Less than 6 per cent (Table IV) of the patients were of risks 3 and 4, because we feel that other methods than spinal anesthesia with metycaine or procain are usually safer for the seriously sick patient. It is of interest to note in the table that as risk increased, dosage decreased.

TABLE IV
RISK*

Dosage Group†	I	II	III	Total
	Per Cent	Per Cent	Per Cent	Per Cent
Risk 1 (good)	45	46	46	46.4
Risk 2 (fair)	42	45	52	47.9
Risk 3 (poor)	11	8	2	5.2
Risk 4 (moribund)	2	1	0	0.5
Total	100	100	100	100.0

* Based on 1346 cases.
† See Table 1.

Efficacy. At the close of each operation the anesthesia was graded by the anesthetist as satisfactory, fair or unsatisfactory. Our aim is to be severe rather than lenient in this grading. For the purpose of this report, anesthesia is considered satisfactory if it extended high enough for the

operation but not too high (that is, so high as to produce embarrassment of breathing, or anesthesia above the clavicles), if it lasted long enough for the operation without being supplemented, if the patient appeared physically comfortable, and if the operating conditions were satisfactory to the surgeon. From Table v it will be seen

Table v
DEGREE OF SATISFACTION

Dosage Group†..	I		II		III		Totals	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Satisfactory.....	155	95.7	462	94.0	597	85.9	1214	90.0
Fair.....	6	3.7	20	4.0	73	10.5	99	7.3
Unsatisfactory...	1	0.6	10	2.0	25	3.6	36	2.7
Total—fair and unsatisfactory..	7	4.3	30	6.0	98	14.1	135	10.0
Totals.....	162	100.0	492	100.0	695	100.0	1349	100.0

† See Table 1.

that in 90 per cent of the cases anesthesia was satisfactory, in 7.3 per cent fair, and in 2.7 per cent unsatisfactory. Satisfaction was most frequent with the small doses, because the demands upon the anesthesia, such as height, duration and freedom from retching, were least in these cases. Vice versa, anesthesia was least satisfactory (85.9 per cent) with the large doses because the site of operation was higher, the operating time longer and the demands on the anesthesia were therefore greater; also because the larger doses in high abdominal operations were much more likely to cause retching of such degree as to trouble the surgeon. Almost all of the large dosage group had upper abdominal operations. If other methods of anesthesia for upper abdominal operations were to be judged by the high standard of relaxation afforded by spinal, no other method that we have seen used could approach a frequency of complete satisfaction as great as 85.9 per cent. For the whole group, the figure of 2.7 per cent for "unsatisfactory" is gratifyingly low.

Of the various causes of dissatisfaction (Table vi) I should regard the drug as

Table vi
CAUSES OF "FAIR" AND "UNSATISFACTORY" IN TABLE v

Dosage Group†..	I		II		III		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Insufficient length	2	1.2	5	1.0	29	4.1	36	2.7
Insufficient height	4	2.5	14	2.8	16	2.3	34	2.6
Too high.....	0	...	0	...	8	1.2	8	0.5
Slow induction...	0	...	3	0.6	1	0.1	4	0.3
Two injections necessary.....	0	...	1	0.2	0	....	1	0.1
Retching, vomiting.....	0	...	7	1.4	36	5.2	43	3.2
Physical distress..	1	0.6	0	...	8	1.2	9	0.9
Totals fair and unsatisfactory	7	4.3	30	6.0	98	14.1	135	10.0

† See Table 1.

responsible for the following only, and then only in part: insufficient length, and retching and vomiting. Faults of technique could be responsible for some of the cases of insufficient length, and several other causes may produce retching and vomiting, such as morphine, and traction on the mesentery. Physical distress outside the operative field, such as pain referred to the shoulder or neck, is obviously a fault of the method not of the drug, and faults of technique are responsible for the other items of insufficient height, excessive height, and rare slowness or failure of anesthesia to develop with the first injection.

The number of cases in which supplementary drugs were used (Table vii)

Table vii
SUPPLEMENTARY ANESTHETICS

Dosage Group†..	I		II		III		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Not supplemented	171	97.2	431	93.5	606	81.6	1208	87.5
Supplementary drug used.....	5	2.8	30	6.5	137	18.4	172	12.5
Total.....	176	100.0	461	100.0	743	100.0	1380	100.0

† See Table 1.

parallels closely the number in which spinal anesthesia was "fair" or "unsatisfactory," but includes also those patients whose preanesthetic narcosis was so poor as to

make the administration of a general anesthetic advisable for its mental effect only. The drugs most frequently used were ethylene, nitrous oxide and cyclopropane. Ether was occasionally added to these and on a very few occasions the rectus muscles were infiltrated with metycaine or with procain. Table VIII shows the reasons why general anesthetics were added.

TABLE VIII
REASONS FOR USE OF SUPPLEMENTARY DRUGS

Dosage Group†	I		II		III		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Insufficient height	3	1.7	10	2.2	12	1.6	25	1.8
Insufficient length	2	1.1	7	1.5	34	4.6	43	3.1
Insufficient depth	..	...	1	0.2	...	....	1	0.1
Retching or vomiting	..	...	8	1.7	44	5.9	52	3.8
Physical distress‡	..	...	1	0.2	14	1.9	15	1.1
Mental (anxiety, etc.)	1	0.6	7	1.5	28	3.8	36	2.6
No record	..	...	2	0.4	14	1.9	16	1.2
Total causes*	6	3.4	36	7.7	146	19.7	188	13.7
Total cases*	5	2.8	30	6.5	137	18.4	172	12.5

* More than 1 cause occasionally present in 1 patient.
† See Table 1.
‡ This refers to discomfort outside of the operative field.

Duration of Anesthesia. Table IX shows the number of operations still going on at the end of certain ten minute periods after the injection of metycaine, and the percentage of these in which spinal anesthesia still persisted. Over 99 per cent still had anesthesia at the end of sixty minutes, 73 per cent after ninety minutes, and 51 per cent after one hundred minutes. This duration of anesthesia is approximately 40 per cent greater than we obtained with procain given by similar techniques and marks *the greatest single advantage of metycaine* over procain. The extra twenty to thirty minutes of anesthesia which metycaine gives have been extremely helpful in such operations as cholecystectomy with choledochostomy, and perineal repair with hysterectomy. Even when anesthesia was of insufficient duration, it wore off so gradually that general anesthesia could be induced without haste before the relaxation of spinal anesthesia disappeared. This also is in very favorable contrast to our experience with procain, for with that drug we have found that muscular rigidity appears almost instantly with the return of sensation, causing embarrassment both to the surgeon and to the anesthetist.

The total at the foot of the table is of special significance: for the whole group anesthesia lasted at least as long as the operation in 97 per cent of the cases.

SIGNS OF IMMEDIATE TOXICITY

Blood Pressure. Changes of blood pressure and the accompanying signs and

TABLE IX
DURATION OF ANESTHESIA

Dosage Group†	I	II	III	Total
For Operations Which Lasted	Anesthesia Outlasted Operation in			
10 minutes	100 per cent (of 171 cases)	100 per cent (of 465 cases)	100 per cent (of 607 cases)	100 per cent (of 1243 cases)
50 minutes	100 per cent (of 32 cases)	100 per cent (of 225 cases)	100 per cent (of 491 cases)	100 per cent (of 748 cases)
60 minutes	100 per cent (of 20 cases)	99 per cent (of 149 cases)	100 per cent (of 399 cases)	100 per cent (of 568 cases)
70 minutes	78 per cent (of 9 cases)	96 per cent (of 96 cases)	98 per cent (of 258 cases)	97 per cent (of 363 cases)
80 minutes	67 per cent (of 6 cases)	90 per cent (of 52 cases)	89 per cent (of 174 cases)	88 per cent (of 232 cases)
90 minutes	50 per cent (of 4 cases)	75 per cent (of 28 cases)	74 per cent (of 96 cases)	73 per cent (of 128 cases)
100 minutes	33 per cent (of 3 cases)	59 per cent (of 17 cases)	49 per cent (of 61 cases)	51 per cent (of 81 cases)
110 minutes	(no cases)	36 per cent (of 11 cases)	21 per cent (of 43 cases)	23 per cent (of 54 cases)
120 minutes	.....	12 per cent (of 8 cases)	8 per cent (of 37 cases)	9 per cent (of 45 cases)
130 minutes	.....	0 per cent (of 1 case)	(no cases)	0 per cent (of 1 case)
Total	99 per cent (of 171 cases)	98 per cent (of 465 cases)	94 per cent (of 607 cases)	97 per cent (of 1243 cases)

This table is to be read, for example, as follows: "For operations which lasted 80 minutes, anesthesia outlasted operation in 67 per cent of 6 Group 1 cases, in 90 per cent of 52 Group II cases, and in 89 per cent of 174 Group III cases, or in a total of 88 per cent of 232 cases."
† See Table 1.

symptoms, such as pallor, nausea, vomiting and depression of respiration, are probably the most commonly watched evidences of immediate toxicity of local anesthetics. In this series blood pressure was recorded in most cases every five minutes, in a few cases only every ten minutes. The greatest drop in systolic pressure occurring at any time during operation is shown in Table x.

Table x
LOWEST SYSTOLIC PRESSURE DURING OPERATION

Dosage Group†	I (106 Cases)	II (261 Cases)	III (249 Cases)	Total (616 Cases)
	Per Cent	Per Cent	Per Cent	Per Cent
Rise of 10 mm. Hg or more*	10	5	2	4.7
Rise or fall of less than 10 mm. Hg*	36	32	15	25.7
Fall of 25 per cent or less*	34	28	27	28.4
Fall of 26 to 50 per cent*	18	29	52	36.5
Fall of 51 to 75 per cent*	1	5	3	3.6
Fall of 76 to 100 per cent*	1	1	1	1.1
Total	100	100	100	100.0

* From preanesthetic level.
† See Table 1.

The changes are shown not in mm. Hg. but in percentage of the preanesthetic level, since this is considered to be more significant.* With small doses of metycaine, the largest group is that showing no drop in pressure (that is, less than 10 mm. Hg.). The same is true of medium sized doses. With large doses, as well as for the total number, the largest group is that showing drops of 26 to 50 per cent. Note that only 1.1 per cent showed extreme drop in pressure at any time during operation, that is, drop of over 75 per cent. A large proportion of these extreme drops were only momentary, occurring during traction on the gall bladder or other upper abdominal organ.

Table xi shows the change in pressure at the close of operation. Nearly half of the cases (48.7 per cent) showed a rise or no drop from the preanesthetic level. Only 0.5 per cent showed pressure lower than 50 per cent of the preanesthetic figure, and none showed a drop greater than 75 per cent.

Table xi
SYSTOLIC PRESSURE AT END OF OPERATION

Dosage Group†	I (101 Cases)	II (248 Cases)	III (232 Cases)	Total (581 Cases)
	Per Cent	Per Cent	Per Cent	Per Cent
Rise of 51 to 75 per cent*	...	1 case	...	0.2
Rise of 26 to 50 per cent*	5	2	1	2.1
Rise of 25 per cent or less*	16	9	9	10.1
Rise or fall of less than 10 mm. Hg*	34	42	31	36.3
Fall of 25 per cent or less*	33	29	36	32.4
Fall of 26 to 50 per cent*	12	17	23	18.4
Fall of 51 to 75 per cent	...	1	1 case	0.5
Fall of 76 to 100 per cent*	...	...	...	0.0
Totals	100	100	100	100.0

* From preanesthetic level.
† See Table 1.

We do not have similar figures for our procain cases, but it is our impression that blood pressure has behaved no worse with metycaine than with procain. Since it appears that the increased potency of metycaine is not accompanied by any increase in vascular depression, the margin of safety of metycaine in this respect seems to be greater than that of procain.

Thoracic Breathing. If the normal expansion of the upper half of the chest with forced inspiration is abolished under spinal anesthesia and is replaced by a sinking-in of the thoracic wall, it means that the effect of the drug on the motor nerves has extended so high that action of the upper intercostal muscles is paralyzed, and that only that of the diaphragm and of the accessory muscles of respiration remains. This condition is apt to be accompanied by insufficient pulmonary ventilation, suboxidation of the blood and therefore of the body tissues generally, and also by a fall in blood pressure. A similar ascending respiratory paralysis occurs under deep general anesthesia.[7] With spinal anesthesia we consider it very important to watch for the presence or absence of thoracic breathing, and to give the patient oxygen if the

* For the preanesthetic level two readings were usually considered: one a few minutes before spinal anesthesia was induced, and the other at the time of the most recent general physical examination. The lower of the two figures was used, since this was considered to represent more nearly the systolic pressure under conditions of physical and emotional calm.

respiratory exchange appears to be seriously diminished. Table XII shows the frequency and duration of paralysis of thoracic breathing. It did not occur with small doses of metycaine but was present in 5.3 per cent of the whole group. Since this table contains a disproportionately small number of small dosage cases, it is to be assumed that the actual incidence was less than 5.3 per cent. It is our impression that this is no greater incidence than in our procain series.

TABLE XII
THORACIC BREATHING

Dosage Group†	I (76 Cases)	II (418 Cases)	III (633 Cases)	Total (1127 Cases)
	Per Cent	Per Cent	Per Cent	Per Cent
Not paralyzed	100.0	96.5	92.9	94.7
Paralyzed 5 minutes	.....	1.9	2.2	2.0
Paralyzed 10 minutes	.....	0.2	2.2	1.3
Paralyzed 15 minutes	.....	0.5	1.4	1.0
Paralyzed 20 minutes	.....	0.2	0.5	0.3
Paralyzed 25 minutes	.....	0.7	.....	0.3
Paralyzed 40 minutes	.....	.....	0.2	0.1
Paralyzed, time not stated	.....	.....	0.6	0.3
Totals	100.0	100.0	100.0	100.0

† See Table 1.

Skin. In most cases alterations from the normal color, moisture and temperature of the skin under spinal anesthesia are probably secondary to drops in blood pressure and to diminution of respiratory exchange. Their importance as evidences of drug toxicity is therefore only secondary. Table XIII shows the incidence and degree of pallor in the cases in which the color of the skin was recorded.

Of 416 recorded cases, the skin became wet in 22, an incidence of 5.3 per cent, and of 413 cases it felt abnormally cool in 9, an incidence of 2.2 per cent.

Since these groups contained disproportionately small numbers of small dosage cases, it is to be assumed also that the figures indicate a considerably greater incidence of pallor, wetness and coolness than would be true for the whole group of 1381 cases.

Retching and Vomiting. The incidence of retching is shown in Table XIV. It increases with the larger doses, and is high for the group as a whole. As was seen in Tables VI and VIII it made anesthesia unsatisfactory in 3.2 per cent, and necessitated the addition of a general anesthetic in 3.8 per cent. Retching is one of the main drawbacks of spinal anesthesia and is not only a function of the drug used but also of the method itself. It may be due to the anesthetic drug, to the preliminary narcotic, to ephedrine, to the emotional state of the patient, and most often to afferent nerve impulses arising in the field of operation (such as from traction on the mesentery) and not ordinarily blocked by spinal anesthesia. As indexes of toxicity and safety of the drug, the effects on blood pressure and on respiration are far more significant.

TABLE XIII
PALLOR

Dosage Group†	I (44 Cases)	II (183 Cases)	III (234 Cases)	Total (461 Cases)
	Per Cent	Per Cent	Per Cent	Per Cent
No pallor	89	80	70	75.7
Slight pallor	7	12	23	16.9
Moderate pallor	2	7	6	6.3
Marked pallor	2	1	1	1.1
Extreme pallor	..	..	..	....
Totals	100	100	100	100.0

† See Table 1.

TABLE XIV
RETCHING

Dosage Group†	I (77 Cases)	II (369 Cases)	III (553 Cases)	Total (999 Cases)
	Per Cent	Per Cent	Per Cent	Per Cent
Retching absent	92	81	62	71.4
Retching present	8	19	38	28.6
Totals	100	100	100	100.0

† See Table 1.

COMPLICATIONS

Complications Occurring during Anesthesia. There were 3 patients who had chills, convulsions or rigidity while they were in the operating room. One of these was a man of forty years with a temperature of 102 degrees F. He was given subdurally 155 mg. of metycaine diluted to 4 c.c. with spinal fluid, and about eight minutes afterwards had a chill lasting three minutes. Operation showed acute appendicitis with local peritonitis, and after operation he developed bronchitis, pyelitis and prostatitis. It is, therefore, fully as likely that the chill was due to the infection as to the spinal injection. On another occasion a woman was given 140 mg. diluted to 4 c.c. with spinal fluid. Immediately after injection she straightened out, turned herself on to her abdomen and remained rigid for ten to twenty seconds. There were no clonic movements and she soon relaxed. An hour later she cried hysterically, so that nitrous oxide had to be given. The rigidity appeared to be hysterical also. The third patient, a man of 6 feet 5 inches, 212 pounds, and fifty-two years of age, was given 80 mg. diluted to 5 c.c. with spinal fluid. Anesthesia extended only to the eleventh thoracic segment. For the operation, proctoscopy, he lay face down, with pelvis elevated. When he was turned on to his back he was pale and his pulse was weak. Oxygen was administered and these signs rapidly disappeared. Two months later he was given the same dose for hemorrhoidectomy. The record does not show what position he was in for operation, but during the operation he had convulsions or chills without changes in color of the skin or in character of the pulse. Convalescence in these latter 2 patients was normal.

A man weighing 215 pounds developed complete respiratory paralysis seventeen minutes after receiving 200 mg. of metycaine. Oxygen was immediately administered by artificial respiration with a gas machine, and the operation of cholecystectomy was continued. Convalescence was complicated by bronchopneumonia. We have had a few other cases of complete respiratory paralysis under spinal anesthesia lasting from ten to thirty minutes with other drugs, and when handled in this way the course in the operating room has been satisfactory.

Postoperative Complications—Source of Data. After each patient's operation, progress and discharge notes are made by the assistant surgeon and by any consultants who may see him. From these, the data of Table xv were compiled. In addition, in about half the cases included in this report, I have read the follow-up notes of the months following discharge from the hospital in order to pick up any late complications that might be due to spinal anesthesia, and have included these in the table. By these two procedures occasional minor complications probably have escaped notation, but it is unlikely that any significant number of major complications occurring during the hospital stay have been omitted. Special effort was made to ferret out neurological complications, whether another anesthetic intervened or not.* Catheterization, vomiting, slight or transient headaches and similar items are not included. Deaths resulting directly from the diseases for which operation was performed without development of complications, are not recorded in this table.

Complications Probably Not Due to Anesthesia. Almost all of the diagnoses listed in Table xv under the heading "Miscellaneous, Probably Not Due to Anesthesia" appear to be complications of the original surgical disease or of the operation, such as intestinal obstruction and infection of wound, or to have occurred coincidentally,

* If a complication occurs in a patient who has been anesthetized more than once, it is considered to be a complication of the most recent anesthesia only, otherwise one death or one complication might be counted two or three times. On this account there are doubtless several deaths that occurred in this series of patients that do not appear in Table xv, because, after the tabulated complication of spinal anesthesia developed, some other anesthetic than metycaine was administered in an attempt to relieve the condition.

TABLE XV
POSTOPERATIVE COMPLICATIONS

	No.	Died
No complication (86.2 per cent)	1188	
No record	14	
Miscellaneous, probably not due to anesthesia		
General (12 cases, 0.9 per cent)		
Fever, unknown origin	6	
Pyemia	2	2 (comp.)*
Scarlet fever	1	
Drug allergy (belladonna)	1	
Bursitis	1	
Tuberculosis of wrist	1	
Related to operative wound (77 cases, 5.6 per cent)		
Infection of wound	48†	2 (comp.)*
Hemorrhage	18	
Rupture of wound	7	
Hernia in scar	3	
Dermatitis (iodine)	1	
Gastrointestinal tract (25 cases, 1.8 per cent)		
Intestinal obstruction	9	1 (or. dis.)‡
Ileus	5	2 (or. dis.)‡
Prolonged vomiting	3	
Dilatation of stomach	2	
Perirectal abscess	2	
Parotiditis	1	
Fecal fistula	1	
Fissure of anus	1	
Stricture of anus	1	
Genitourinary tract (42 cases, 3.0 per cent)		
Pyelitis, pyelonephritis, nephritis	17	
Cystitis	13	
Ureteral colic	3	
Anuria	2	
Urinary fistula	2	
Epididymitis	2	
Obstruction of ureter	1	
Urethritis	1	
Furuncle of labium majus	1	
Abdominal cavity (9 cases, 0.7 per cent)		
Peritonitis	6	5 (comp.)*
Pelvic abscess	3	
Respiratory (33 cases, 2.4 per cent)		
Atelectasis	8	2 (comp.)*
Pneumonia	6	
Pharyngitis, tonsillitis	5	
Bronchial asthma	5	
Bronchitis	4	
Head cold	2	
Pulmonary edema	2	
Pleurisy	1	
Circulatory (36 cases, 2.6 per cent)		
Embolus of lung	10	4 (comp.)*
Shock (see Table XVI)	9	1 (comp.)*
Phlebitis	6	
Auricular fibrillation	3	
Coronary occlusion	2	2 (comp.)*
Cardiac failure	2	
Embolus of extremities	2	
Thrombosis of mesentery	1	
Epistaxis	1	
Psychiatric (6 cases, 0.4 per cent)		
Psychosis	5	
Delirium	1	
Neurological (25 cases, 1.8 per cent)		
Headache	6	
Weakness of lower extremities	6	
Hiccup	5	
Hemiplegia	2	1 (comp.)*
Hyperesthesia	2	
Numbness	2	
Anesthesia (thigh)	1	
Coccygodynia	1	
Unclassified (0.2 per cent)		
Backache	3	

* Died of the complication.
† Many of these were of operations into infected areas, such as excision of rectum.
‡ Died of original disease.

such as bursitis. In some, for example prolonged vomiting, it is possible though not probable that anesthesia could have played a contributing part. The cases of fever of unknown origin are considered not due to anesthesia, since they showed no localizing signs.

Respiratory, Circulatory, and Psychiatric Complications. In the etiology of the respiratory, circulatory and psychiatric complications, the anesthetic drug and method may or may not have had a part. This could only be vaguely estimated from large parallel series of cases anesthetized by various drugs and methods. We have found pulmonary complications to depend more upon the site and duration of operation, the age of the patient, and the presence of infection anywhere in the body than upon the anesthetic drug or method. Shock of minor degree is not recorded as a complication, because, like a mild febrile reaction, it is expected to occur frequently after many operations and because prophylactic and therapeutic transfusions were very commonly given at the close of operation when it was thought that severe shock might otherwise develop. Significant details of the 9 patients that did develop severe shock are given in Table XVI. The one who died was in very poor condition before operation, and the operation was such as to cause severe shock. In 2 cases the anesthesia was thought to be a contributing cause.

Neurological Complications. Of all postanesthetic complications, these are the ones to which most interest attaches in evaluating a new drug for spinal anesthesia. Headache of sufficient severity to be noted on the record occurred in 6 patients, an incidence of 0.4 per cent. It is quite possible that there were other cases of severe headache that were not recorded. Of the 6, 3 were considered neurasthenic or psychoneurotic before or after anesthesia because of the multiplicity and diversity of their complaints, and in 5 the pain was definitely relieved by lying with the head low. This symptom, therefore, seemed to depend on a neurotic constitution or on the fact of lumbar puncture, rather than on any possible irritative property of the drug used. There was no case of severe long-

persisting headache, and none in which spinal fluid drainage seemed indicated.

Anesthesia of a limited area of the left thigh occurred in one patient following panhysterectomy; sensation was nearly normal at the time of discharge from the hospital fifteen days later.

Hyperesthesia of the left thigh occurred in one patient following resection of the sigmoid and left oophorectomy. It had disappeared when she was seen again after three months. I have observed several similar abnormalities of sensation of the thighs when other drugs, including procain, have been used, and in every case the operation had been pelvic. It therefore seems fully as likely to be due to operative trauma as to spinal anesthesia. Another patient had an unusual hyperesthesia before operation. A normal appendix was removed. During spinal anesthesia she complained of hyperesthesia at the upper border of the zone of anesthesia. Troublesome hyperesthesia of the upper part of the trunk persisted. Three years later studies including spinal fluid analysis and dynamics were made by the neurological consultant, who corroborated the earlier diagnosis of psychoneurosis as the explanation of the complaint.

One patient complained of transient numbness of the left arm on the second day after spinal anesthesia. Another patient, a woman aged forty-six years, was given 240 mg. of metycaine for cholecystectomy. This was followed in twelve days by subtotal thyroidectomy under cyclopropane. There was no complaint until ten months later, when she developed numbness of the feet and legs which caused some difficulty in walking. The consulting internist found no other neurological signs except absence of bone conduction in the legs. A month later examination was likewise negative, but the sense of numbness had risen to the trunk. It was not considered due to spinal

TABLE XVI

SHOCK

Case	Preoperative Condition	Risk*	Operation	Cause of Shock	Outcome
No. 1..	Ulcerative colitis	2	Resection of rectum and sigmoid	Operation	Recovered
No. 2..	Cholelithiasis	2	Cholecystectomy, choledochostomy, appendectomy	Operation	Recovered
No. 3..	Ulcerative colitis; syphilis; obesity; secondary anemia; faint heart sounds	3	Colectomy, first stage	Poor preoperative condition; operation	Recovered
No. 4..	Hemorrhoids	1	Hemorrhoidectomy	Too much preliminary narcosis; anesthesia	Recovered
No. 5..	Carcinoma of prostate; cardiac decompensation; B.P. 230/120	3	Suprapubic prostatectomy	Poor preoperative condition; operation; hemorrhage	Recovered
No. 6..	Carcinoma of bladder; marked emaciation and anemia; cardiac decompensation; weak	3	Cystectomy, hysterectomy	Poor preoperative condition; operation	Died; surgical shock, pulmonary edema, auricular flutter
No. 7..	Carcinoma of rectum	1	Colostomy	Too much preliminary narcosis; anesthesia	Recovered
No. 8..	Hydronephrosis	2	Nephrectomy	Loss of blood during operation	Recovered
No. 9..	Cholelithiasis	2	Cholecystectomy, choledochostomy	Cardiac infarct	Embolus to arm; embolectomy, amputation; recovered

* See Table IV.

anesthesia. Three and a half years after the operation she was seen for thyroid check-up; in the interim she had apparently recovered completely from the sensory disturbance, which remained undiagnosed.

Three patients had postanesthetic backache. One of these complained of backache before operation and was regarded as psychoneurotic at that time. The operation was merely dilatation and currettage. The backache was worse after operation, but was much diminished when she was seen nine months later for another complaint. Another developed backache following hysterectomy; the orthopedic consultant believed the pain was dependent on postoperative disuse, which, in turn, resulted from fear. In the third case the use of the lithotomy position for a patient with pre-existing severe lumbar lordosis was thought to be the likely cause of the pain. In none of these 3 cases was spinal anesthesia considered to be the probable cause of trouble by the surgeons or consultants.

One patient who had a repair of cervix and of perineum was considered to have an anxiety neurosis while she was still in the hospital. Six weeks later she complained of pain and tenderness in the region of the coccyx. This gradually gave way to stiffness of the neck and headache and these, in turn, to dysmenorrhea and lumbar backache. These latter symptoms were considered due to retroversion of the uterus. None of the several physicians who saw her even suggested that any of her symptoms might be due to spinal anesthesia.

There were 6 patients among the 1381 who developed weakness of the lower extremities following spinal anesthesia. The chief neurosurgeon of the Clinic, Dr. Gilbert Horrax, has reviewed the condensed histories of all, and his conclusions are given for each case. Some of these show a type of nerve injury that could very well be due to extreme plantar flexion caused by tightness of the bedclothes over the feet while the patient lay supine in bed, still relaxed by spinal anesthesia or by postoperative analgesics. This is a phenomenon that has been observed sufficiently often to be well recognized as a possible sequel to general as well as to spinal anesthesia.

In 1 of these 6 patients no suspicion rested on the anesthesia as a possible causative factor. The patient, a woman of fifty-four years, had a cholecystectomy under spinal anesthesia (150 mg. metycaine) and twenty-one days later her wound ruptured and was resutured, again under spinal (100 mg.). Intestinal obstruction developed which was relieved by laparotomy fourteen days later. Her condition was so poor at this time that ethylene intratracheally and metycaine field block were used instead of spinal anesthesia. Fifty days after the first operation she began to get up, and fifteen days later noticed that her legs were weak. The neurological consultant believed the trouble was due to her prolonged severe sickness and to confinement in bed. Steady improvement followed.

The second patient, who had been given 50 mg. of metycaine for hemorrhoidectomy, noted weakness of the left leg when she began to get out of bed one week after operation. Dr. Horrax found definite weakness of all movements of the left leg and foot but no changes in reflexes or sensation. When next seen, four months later, the weakness had apparently disappeared. Ten months after operation she developed a definite psychoneurosis. The weakness could have been from a lesion of the cauda equina or of the lower part of the cord, or it could have been purely functional, dependent upon the psychoneurosis.

In the third case the data are too meagre to permit the drawing of any conclusion concerning diagnosis or etiology. The patient was given 160 mg. of metycaine for interposition operation and perineorrhaphy. After operation "she complained of some weakness of the left leg, but this cleared up" before she left the hospital. When she was seen four months, eight months, and again twenty months after operation, the leg was giving her no trouble.

The same dose was given to the fourth patient for cholecystectomy and appendectomy and no note was made of any trouble while she was in the hospital. Five weeks after operation she complained of numbness and hypoesthesia of the lateral aspect of the right leg and some paralysis of the right peroneal muscles, which were moderately bothersome to her in walking. The condition at that time, however, was definitely improving and complete recovery occurred some time before her return for thyroidectomy five months later. This appears to have been a transient lesion in the cauda equina or at a point peripheral thereto, with spinal anesthesia or trauma from bedclothes as the probable cause.

The fifth patient was given 180 mg. of metycaine for cholecystectomy and choledochostomy. After operation he ran a low fever of undetermined origin, which was still present when he left the hospital nineteen days later. One month after operation he complained of numbness and slapping of the left foot, and when seen one week later by the orthopedic consultant there was weakness but not paralysis of the left anterior tibial and peroneal muscle group. Ten weeks after operation the same condition was present, and no atrophy had developed. When seen again four months after operation he had completely recovered in the interval. Three years later he wrote that at the age of sixty-three years he was playing up to six sets of tennis a day. His trouble could have been due either to a lesion of the cauda equina or to trauma from bedclothes.

The sixth patient was discharged from the hospital without complaint the day after excision of a rectal polyp under 75 mg. of metycaine. Two months later there was toe-drop, but the reflexes were normal. Four months after operation there was no evidence of toe-drop or other muscular weakness. This also could have been caused either by trauma from bedclothes or by a lesion of the cauda equina.

In summary of these 6 cases of weakness, it appears that in the first the trouble was due to prolonged invalidism, in the third no conclusion can be drawn, in the second the causative factor could have been either spinal anesthesia or psychoneurosis, and in the other 3 either spinal anesthesia or trauma from bedclothes. In no case does the weakness appear to have been due definitely or exclusively to spinal anesthesia. Complaint of the symptom was made from a few days to two months following operation, but information is not available as to whether the weakness was noted when each patient first began to walk. In all cases the weakness had entirely cleared up in less than six months.

SUMMARY AND CONCLUSIONS

Metycaine is more potent than procain and clinically appears to be no more toxic. The duration of spinal anesthesia with doses of metycaine ranging from 40 to 200 mg. has been 40 to 50 per cent longer than we have obtained with procain. This has been found to be a decided advantage, especially for gall-bladder operations. Other advantages of the drug are that it gives more prompt, more intense and longer anesthesia by the methods of local infiltration, field block and nerve block, that it is effective by topical application to the eye and to the urethra, and that solutions of it are to a degree self-sterilizing.

The technique of administration is given in detail and 1381 cases are analyzed in regard to age and risk of patient, type of operation, dosage of metycaine, efficacy and duration of anesthesia, behavior of circulation and of respiration, occurrence of retching and vomiting and other undesirable phenomena during anesthesia, and postoperative complications.

There have been no deaths in our series attributable to spinal anesthesia, and few, if any, postoperative complications for which it is surely or solely to blame. Brief histories are given of the cases of postoperative neurological complications. Suspicion points rather strongly at the anesthesia as the likely cause of 4 cases of transient footdrop, but in one of these a

psychoneurosis may have been to blame, and in the others there may have been nerve injury from tight bedclothing.

Because of its greater power and longer duration of anesthesia, apparently without greater toxicity, we much prefer metycaine to procain for spinal, regional and local anesthesia. It seems to us almost certain, however, that further progress in drugs for spinal anesthesia will be toward those that are effective in far smaller amounts or weaker dilutions. Thus at the present time we are using tetracain (pontocaine) with glucose[11] for the majority of spinal anesthesias, and nupercain (1:1500) for operations of very long duration, because it is our distinct impression that circulation and respiration are better maintained and that retching is less frequent with these drugs than with either procain or metycaine.

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SUBMUCOUS LIPOMAS OF COLON AND RECTUM

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EXCLUDING adenomatous polyps, lipomas are the most common benign tumors of the gastrointestinal tract. They may be divided into two types, according to the site of their origin: (1) the submucous, and (2) the subserous. Comfort, in 1931, reported a series of 181 submucous lipomas of the gastrointestinal tract; 92 were situated in the large intestine and rectum, and 75 had produced symptoms. He concluded that, in the large intestine, the cecum, ascending colon and sigmoid flexure were the most common sites of origin of submucous lipomas, in the order named.

Figures on the incidence of submucous lipomas of the colon vary in each individual clinic, depending on the type of material studied and the number of necropsies obtained. Kirshbaum, at the Cook County Hospital, found only 2 such tumors among 5734 consecutive necropsies. In Comfort's series of 3924 consecutive necropsies there were 24 submucous lipomas of the entire gastrointestinal tract, 11 of which were in the colon and rectum, an incidence of about one in 356 cases in which the patient came to necropsy. Apparently they are more common than one would surmise but the number of these growths which produce symptoms and necessitate surgical intervention is rather small.

A case which was seen recently at The Mayo Clinic stimulated our interest sufficiently to make a study of other cases that had been seen here and to review other cases which had been reported.

Comfort, in his exhaustive study, reported 3 cases with symptoms which had been seen at the Clinic up until 1928, and we are adding 3 more seen since 1928, making a total of 6 cases. Reports of 91 other cases in which the patient had symptoms were found in the literature, making a total of 97 cases with symptoms. Nineteen cases in which the condition was found at necropsy, and which have been reported in the literature, have been added, making a total of 116 cases.

REPORT OF CASES

CASE 1. A white man, aged fifty-one years, who was a broker and married, entered the clinic October 14, 1932. An appendectomy had been performed nineteen years before. Nine years before he came to the clinic he had had an attack of acute pain in the lower abdomen and followed by the formation of a mass in the left lower abdominal quadrant. He had been ill for five or six days, and after he had passed something "that looked like a part of a bicycle inner tube," the mass had disappeared and he had obtained relief. However, for the next year and a half he had felt weak and "run-down." He had been constipated ever since and daily doses of mineral oil had been required to obtain a bowel movement. For the past year he had had slight, short cramp-like abdominal pains, chiefly in the right lower abdominal quadrant, lasting only a few minutes at a time; they had not been accompanied by nausea, vomiting, diarrhea or bloody stools but he had noted some abdominal distention. There had not been any loss of strength or weight.

Physical examination revealed a small man who weighed 115 pounds (52.1 kg.). The value for the blood pressure, expressed in millimeters of mercury, was 130 for the systolic and 90 for the diastolic. The pulse rate was 76, and the temperature 98.4 degrees F. Results of the general examination were negative except for slight generalized abdominal distention and tenderness. No masses could be palpated. Examination of the blood revealed that the result of the Kahn test was ± and that of the Kline also was ±. A blood count did not reveal

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any abnormality. The value for the urea was 28 mg. per 100 c.c. of blood.

The result of proctoscopic examination was negative and examination of the stools did not reveal any parasites or blood. The urine was normal. Roentgenologic examination of the thorax revealed old tuberculosis at the upper part of both lungs, but examination of the sputum did not reveal Mycobacterium tuberculosis. Roentgenologic examination of the colon revealed a multilobular mass at the right colonic flexure and a tendency to intussusception (Fig. 1).

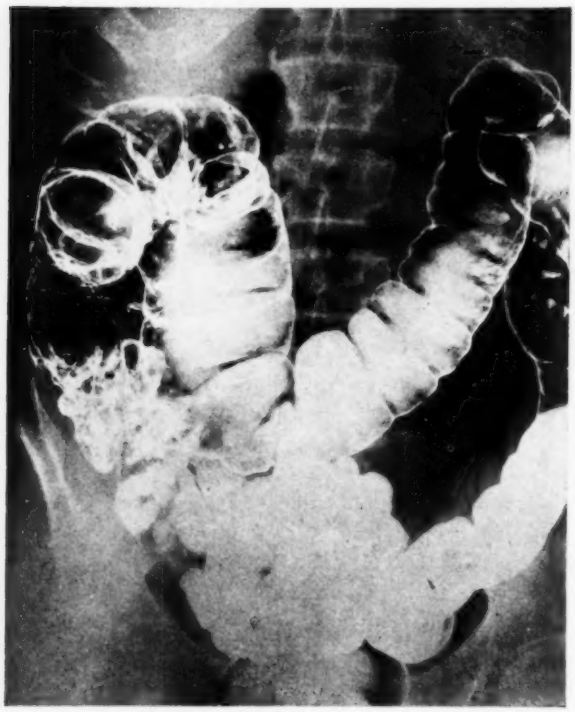

Fig. 1. Multilobular polypoid mass at the right colonic flexure, with a tendency to intussusception.

Vaccne was administered intraperitoneally and the patient was operated on under combined spinal and inhalation anesthesia. Exploration of the abdomen through a right rectus incision revealed a soft, multilobular mass, apparently a lipoma, which was about 5 by 7 cm. in size and situated in the hepatic flexure of the colon. There was no evidence of malignancy, but many adhesions remained from the previous operation. The involved loop of colon was brought outside of the abdomen without interfering with the blood supply, as the first stage of a Mikulicz resection.

Ten days later 8 cm. of colon which contained the growth was removed with the cautery (Fig. 2). The pathologist reported a submucous polypoid lipoma which measured 6 by 7 by 5 cm. Clamps were applied to the spur on two occasions. Finally, about two months after the original operation the bowel was separated down to the peritoneum without opening the peritoneal cavity, and was closed with three rows of chromic catgut. One Penrose drain was inserted down to the fascia.

Fig. 2. Submucous polypoid lipoma (gross specimen removed at operation).

Three months later the patient reported that he was in excellent condition.

Case II. A merchant, aged fifty-eight years, came to the clinic on February 22, 1934, because of diffuse abdominal distress of three months' duration. His father had died of an intestinal disorder at the age of sixty-four years; the patient did not know the exact nature of the disorder. The patient had undergone a tonsillectomy twelve years previously.

For three months before he came to the clinic he had had attacks of diffuse abdominal pain which had been colicky in character and had lasted from a few minutes to several hours. The pain had not been related to the quality or quantity of food, and there had not been any vomiting, diarrhea or bloody stools. A roentgenologic examination of the gastrointestinal

tract had been made elsewhere, and a suspicious prepyloric lesion had been diagnosed. Gastric analysis had revealed an achlorhydria and the patient had been given a special diet and hydrochloric acid by mouth. For two weeks before he came to the clinic the pain had been localized in the left side of the abdomen; examination of the stools had revealed occult blood. He had lost some strength and 9 pounds (4.1 kg.) since the onset of his illness.

Physical examination at the clinic revealed a well preserved man who had hypertension. The value for his blood pressure, expressed in millimeters of mercury, was 210 for the systolic and 110 for the diastolic. The pulse rate was 80 and the temperature was 99.2 degrees F. His weight was 176 pounds (79.8 kg.). There were no palpable abdominal masses. Roentgenologic examination of the stomach did not reveal any intrinsic lesion but did disclose a small para-esophageal diaphragmatic hernia. Roentgenologic examination of the colon revealed a polypoid lesion, 3 by 3 cm., at the splenic flexure. Examination of the blood and urine did not reveal any abnormality.

He refused to submit to operation but returned home. With the use of a semisolid diet, mineral oil, olive oil enemas, and belladonna, he remained comfortable, but returned to the clinic following another attack.

Roentgenologic examination of the colon again revealed a lesion about 3 by 3 cm., which was situated high in the descending colon, just distal to the splenic flexure.

Vaccine was administered intraperitoneally and an exploratory laparotomy was performed by Dr. C. F. Dixon, under combined spinal and inhalation anesthesia. In the first part of the descending colon a questionably malignant polypoid mass was felt. Examination of the liver did not reveal metastasis. An exteriorization operation was performed with difficulty; about 14 cm. of the splenic flexure and the descending colon was removed. The pathologist reported a sessile submucous lipoma, which measured 3 by 3 by 2.5 cm. (Fig. 3). The proximal loop of the colon was punctured with a cautery the next day. At the second stage of the operation, the colonic stoma was closed over a large rectal tube.

According to a letter from his wife, the patient was in good health seven months later.

Case III. A married woman, aged sixty-five years, entered the clinic May 16, 1935, because of abdominal distress which had been present for five weeks. She had had the usual diseases of childhood. The family history was unimportant. She had been married twenty-four years; her husband was living and well, and there were five children who were living and well. She never had had a miscarriage. Her menstrual history was not abnormal; the menopause had occurred at the age of fifty years.

Fig. 3. Sessile submucous lipoma; specimen removed at operation.

Three years before she came to the clinic she had had an attack of drowsiness and sleepiness, but had not been unconscious. At this time her family physician had discovered that she had diabetes mellitus. She had been given a strict diet for eight months and insulin had been administered; the largest dose had been 10 units, three times a day. Since then, her diet had not been very rigid, but the quantity of carbohydrates had been limited. She had tested her own urine once a week, and occasionally had taken 5 units of insulin, which had kept the urine free of sugar.

From 1920 to 1925 she had had attacks of distention and a feeling of fullness after meals

which were relieved by taking bicarbonate of soda. There had not been any other gastrointestinal symptoms and she had enjoyed very

Fig. 4. Large polypoid lesion in the ascending colon, near the hepatic flexure, with a definite tendency toward intussusception.

good health until the onset of her present illness. Five weeks before she came to the clinic she had begun to experience attacks of sharp, short, colicky abdominal pain, more severe in the left side of the abdomen. The attacks had occurred in the afternoon and had continued until 10 p.m., when she had obtained relief. This episode had lasted about ten days and had subsided. She had noticed some constipation, but there had not been any vomiting. Two weeks before her registration at the clinic she had had a more severe attack, with abdominal distention, nausea and vomiting. One week later she had had another attack; the pain had extended to the epigastrium and left hypochondrium. She had noted that the constipation was increasing. She never had noted blood in the stools. The day she came to the clinic she had had another attack, which had been characterized chiefly by vomiting. She had vomited five times during the day and had not had any bowel movement. There had been a slight loss of weight during the past five weeks.

Physical examination revealed a moderately obese woman who was 5 feet ¾ inch (154.4 cm.) in height and weighed 163 pounds (74 kg.). Slight pallor of the skin and mucous membranes, sclerosis, grade 1 of the peripheral vessels, and slight senescent arthritis of the fingers were noted. The value for the blood pressure, expressed in millimeters of mercury, was 148 for the systolic and 82 for the diastolic. The pulse rate was 100 beats per minute and the temperature was 99.2 degrees F.

Examination of the head, neck and thorax did not reveal any abnormality. There was some abdominal distention, but no masses were felt and no tenderness was elicited. Rectal and pelvic examinations did not reveal anything abnormal. There were 4,620,000 erythrocytes and 7600 leucocytes in each cubic millimeter of blood. The value for the hemoglobin was 14.1 gm. per 100 c.c. of blood. Three hundred cubic centimeters of urine contained 3.75 gm. of sugar; no acetone or diacetic acid was found. The stools did not contain blood. The value for the sugar was 184 mg. per 100 c.c. of blood and that for the urea was 14 mg. per 100 c.c. of blood. The Wassermann reaction on the blood was negative. Gastric analysis disclosed that the value for the total gastric acidity was 52, and that the value for the free hydrochloric acid was 48, according to the method of Töpfer. Proctoscopic examination of the distal 24 cm. of the rectum and roentgenologic examination of the stomach failed to reveal any abnormality. An ordinary roentgenogram of the abdomen disclosed hypertrophic changes in the lumbar vertebrae and phleboliths on both sides of the pelvis. A roentgenogram of the thorax revealed calcification of the arch of the aorta. A teleoroentgenogram disclosed that the diameters of the cardiac shadow were as follows: R, 6 cm.; L, 8 cm., and C, 27 cm. Examination of the ocular fundi revealed slight generalized retinal arteriosclerosis. Cystoscopic examination revealed a slight chronic urethritis, which was not considered important as the cultures were negative.

Roentgenologic examination of the colon revealed a large polypoid lesion in the ascending colon, near the hepatic flexure, and a definite tendency toward intussusception (Fig. 4).

A diagnosis of colonic obstruction was made and the patient was prepared for operation. Regulation of the diet and administration of insulin produced an improvement in her general condition. Vaccine was administered

intraperitoneally and operation was performed on May 24, 1935. Under combined spinal and inhalation anesthesia, a right rectus incision was made. This revealed a tumor of the ascending colon, which was situated just below the hepatic flexure. The tumor arose from the mesenteric border and extended into the lumen of the intestine and as it felt like a lipoma it was decided to do a Mikulicz resection. The loop of bowel which contained the tumor was brought to the outside as the first stage of the procedure.

The patient reacted well to this procedure. Sugar appeared in the urine in greater quantities and the value for the blood sugar increased to 227 mg. per 100 c.c. As a result of the use of insulin and the administration of fluids intravenously and subcutaneously, her condition progressed satisfactorily. On the fourth day after the operation the proximal loop was punctured with a cautery and she began to take fluids by mouth. On the tenth day she received a restricted diet.

On June 4, 1935, the second stage of the Mikulicz operation was performed; the loop of intestine which contained the growth was removed with cautery; a clamp was left on the spur. The pathologist reported the specimen to be 8 cm. of the hepatic flexure of the colon, which contained a submucous lipoma, 5 by 4 by 4 cm.

She had a slight reaction characterized by fever and chills, the first day after the latter operation but, otherwise, the convalescence was uneventful. The clamp was tightened daily until it came off on the eighth day. Another clamp was applied June 25, 1935; this was tightened daily until it came off on the ninth day. She was dismissed from the hospital and sent home.

She returned for closure of the colonic stoma on August 2, 1935. This was done by using three rows of sutures of chromic catgut and then reinforcing the suture line by bringing over the aponeurosis. When she was dismissed from the hospital on August 22, 1935, she was in excellent condition.

COMMENT

Clinically, submucous lipomas of the colon and rectum may be divided into two groups: (1) those that produce symptoms (Table I), and (2) those that do not produce symptoms (Table II).

In 101 of the entire series of 116 cases (Table III), the age of the patient was noted. The youngest patient was sixteen years and the oldest patient was eighty-three years of age; the average age of these patients was 49.8 years.

The age of the patient was noted in 86 of the 97 cases in which symptoms occurred; the youngest was sixteen years and the oldest patient was eighty-three years of age; the average age of these patients was forty-seven and one-half years.

The age of the patient was noted in 15 of the 19 cases in which symptoms were absent. The youngest patient was thirty-five years and the oldest patient was eighty-three years of age; the average age of these patients was sixty-three years.

It seems then in the cases in which symptoms are present the lipoma occurs at a younger age than it does in cases in which symptoms are absent. In 56 cases, or about 65 per cent of the cases in which symptoms occurred and in which the age of the patient was stated, the tumor occurred between forty and sixty years. This is of interest because these are the decades in which carcinoma is most likely to occur.

In the 103 cases in which the sex was noted, 41 of the patients were men and 62 were women (Table III). In the cases in which symptoms were present, 35 of the patients were men and 51 were women, while in the cases in which there was an absence of symptoms, 6 of the patients were men and 11 were women. It appears then that lipomas of the colon and rectum are more common among women than they are among men.

There is no symptom complex that is diagnostic of a lipoma of the colon or rectum. Cases of lipoma of the colon may be divided into two groups, according to the clinical course. In the first group, symptoms have been present for many years. There have been attacks which have lasted for a few minutes, for many hours, or for days. After the subsidence of these attacks, the patient enjoys good health

TABLE I

CASES IN WHICH SUBMUCOUS LIPOMAS OF THE COLON OR RECTUM PRODUCED SYMPTOMS

Author and Date	Age in Years and Sex	Situation of Lipoma	Duration of Symptoms	Pain	Constipation	Diarrhea	Blood in Stools	Loss of Weight	Anemia	Palpable Mass	Positive Roentgenologic Sign	Treatment	Remarks and Results
Bauer, 1757[48]	M	Possibly colon	Seven weeks	+	+	..	..	..	..	..	..	.....	Passed spontaneously; recovered
Meckel, 1818[48]	..	Colon	.....	..	..	..	..	..	..	..	..	.....	Specimen
Sangalli, 1860[48]	..	Descending colon	.....	..	..	..	..	..	..	..	..	.....	Specimen
Castelain, 1870[48]	43 M	Probably colon	Four weeks	+	+	..	+	..	..	..	..	.....	Passed spontaneously; recovered
Hoyt, 1872[48]	55 F	Probably colon	.....	..	+	+	..	..	..	+	..	Anal removal	Died
Afezou, 1875	83 F	Rectum	Years	+	+	+	..	..	..	..	..	.....	Passed spontaneously; recovered
Voss, 1880[48]	47 F	Sigmoid flexure	.....	..	..	..	..	..	..	+	..	Anal removal	Recovered
Albrecht, 1880[48]	51 M	Colon	Six weeks	+	..	+	+	+	..	..	..	.....	Passed spontaneously, recovered
Esmarch, 1882[48]	..	Sigmoid flexure	.....	..	..	..	..	..	..	+	..	Anal removal	Recovered
Esmarch, 1882[48]	..	Sigmoid flexure	.....	..	..	..	..	..	..	+	..	Anal removal	Recovered
Tuffier, 1882[48]	43 F	Sigmoid flexure	Nine months	+	+	..	..	..	..	+	..	Colostomy	Died
Tédenat, 1885[48]	64 F	Rectum	Six years	+	+	+	+	..	..	..	..	Anal removal	Recovered
LaGarde, 1886[48]	65 M	Rectum	Three years	+	+	..	+	..	..	+	..	Anal removal	Recovered
Link, 1890	45 M	Descending colon	Five years	+	+	..	+	..	..	+	..	Anal removal	Recovered
Hofmokl, 1892[48]	39 F	Ascending colon	.....	..	..	..	..	..	..	..	..	Resection	Died
Richardson, 1892	52 M	Cecum	Forty years	..	..	..	..	..	..	..	..	Removal	Died
Durante, 1896[34]	40 M	Cecum	.....	..	..	..	..	..	..	..	..	Local excision	Recovered
Brunner, 1899	51 M	Rectum	Five days	+	+	..	+	..	..	+	..	Clostomy and resection	Recovered
Bodenhamer, 1898	37 M	Rectum	Sixteen years	..	..	..	..	..	..	+	..	Anal removal	Recovered
Bierring, 1898[48]	23 F	Possibly colon	Years	+	+	..	..	..	..	..	..	.....	Passed spontaneously; recovered
Russell, 1899	56 M	Sigmoid flexure	Twenty-three years	+	+	..	+	..	..	+	..	Laparotomy and removal	Recovered
Heurtaux, 1900[48]	60 F	Descending colon	.....	..	..	..	..	..	..	+	..	Anal removal	Recovered
Bland-Sutton, 1900	44 M	Ascending colon	Seven years	+	+	..	..	..	..	+	..	Laparotomy and removal	Recovered
Michaux, 1900	56 F	Descending colon	.....	..	..	..	..	..	..	+	..	Colostomy	Died
Gross, 1900	47 M	Transverse colon	Fourteen days	+	..	..	..	..	..	+	..	Laparotomy and removal	Recovered
Langemak, 1900	57 F	Cecum	Nine years	+	..	+	..	..	..	..	..	Laparotomy and resection	Recovered
Gant, 1902[48]	..	Rectum	.....	..	..	..	..	..	..	+	..	Anal removal	Recovered
Tuttle, 1902	..	Rectum	.....	..	..	..	..	..	..	+	..	Anal removal	Recovered
Tuttle, 1902	..	Rectum	.....	..	..	..	..	..	..	+	..	Anal removal	Recovered
Haasler, 1902	25 M	Transverse colon	Years	+	..	+	+	..	..	+	..	Resection with Murphy button	Recovered
O'Conor, 1903	45 F	Cecum	.....	+	+	..	..	+	..	+	..	Laparotomy and removal	Recovered

Ward, 1904	29 F	Cecum	One year	+	+	..	..	..	..	+	..	Laparotomy and removal	Recovered
Hellström, 1906	47 M	Transverse colon	Two years	+	+	..	..	..	..	+	..	Laparotomy and removal	Recovered
Hellström, 1906	16 F	Colon	.....	..	..	..	..	..	..	..	..	.....	Passed spontaneously; recovered
Hellström, 1906	47 M	Colon	Eight years	+	+	+	..	..	..	+	..	.....	Passed spontaneously; recovered
Knowlton, 1906	56 F	Cecum and ascending colon	Twenty years	+	+	..	..	..	..	+	..	Laparotomy and resection	Recovered
Lorenz, 1906[11]	58 M	Cecum	.....	+	+	..	..	+	..	+	..	Resection	Recovered
d'Este, 1906	54 M	Ascending colon	Two months	+	+	+	..	..	..	+	..	Resection	Recovered
Hochenegg, 1909[48]	..	Cecum	.....	+	+	..	..	..	..	+	..	Resection	Recovered
Klein, 1909	40 F	Transverse colon	Two months	+	+	..	..	..	..	+	..	Resection	Recovered
Stetten, 1909	20 F	Transverse colon	Three months	+	+	..	..	..	..	+	..	Resection	Recovered
Mattes, 1909[11]	76 M	Sigmoid flexure	Three weeks	+	+	+	+	..	..	+	..	Resection	Died
Westbrook, 1910[11]	34 F	Ascending colon	One year	+	..	+	..	..	..	+	..	Resection	Recovered
Finney, 1911[58]	35 M	Descending colon	Five weeks	+	..	..	+	..	..	+	..	Resection	Recovered
Petit and Malapert, 1912	32 F	Rectum	.....	..	+	..	+	..	..	+	..	Local removal	Recovered
Fantino, 1912[11]	37 M	Transverse colon	Three days	+	+	..	..	..	..	..	..	Colostomy and local excision	Died
Cassanello, 1912	51 M	Sigmoid flexure	Years	..	+	..	+	..	..	+	..	Resection	Recovered
Hengstenberg, 1913[11]	21 F	Descending colon and sigmoid flexure	.....	+	..	..	..	..	..	..	..	Resection	Died
Cicala, 1913[11]	59 F	Cecum	.....	+	..	..	..	..	..	+	..	Local excision	Recovered
Collins, 1913	50 F	Cecum	Two weeks	+	..	+	+	..	..	+	..	Removal	Recovered
Kellert, 1917	46 M	Cecum	Six months	+	+	..	..	+	..	+	..	Ileocolostomy and resection	Recovered
Lewisohn, 1918	50 F	Ascending colon	Two months	+	..	..	..	..	..	+	+	Resection and lateral anastomosis	Recovered
Nigst, 1918[11]	55 M	Descending colon	One month	+	+	+	..	..	..	+	..	Resection	Recovered
Summers, 1918	31 F	Cecum	Two weeks	+	+	..	..	..	..	+	+	Local excision	Recovered
Landauer, 1919[11]	59 M	Ascending colon	Seventeen months	+	+	..	..	..	..	+	..	Local excision	Recovered
Kothny, 1920	43	Large intestine	Three months	+	..	..	..	+	..	+	..	.....	Passed spontaneously; recovered
Longo, 1920[11]	47 F	Cecum	Forty-seven days	+	+	..	..	..	..	+	..	Local excision	Recovered
Labey, 1920	46 F	Ascending colon	Four years	+	+	..	+	..	..	..	+	Cecostomy and resection	Recovered
Greenwood, 1920[2]	31 F	Ascending colon	.....	..	..	..	..	..	..	..	..	Resection	Recovered
Compton, 1920[3]	30 F	Cecum	.....	..	+	..	..	..	..	..	..	Resection	Recovered
Forster, 1921[11]	59 F	Transverse colon	Ten years	+	..	..	..	..	..	+	..	Resection	Recovered
Carlucci, 1921	43 F	Transverse colon	Three months	+	+	..	+	..	..	..	..	Resection	Died
Chalier, 1921	46 M	Descending colon	Five days	..	+	..	+	..	..	..	..	Local excision	
Brouwer, 1921	45 M	Ileocecal valve	Four years	+	+	..	..	..	..	+	..	Removal	
Cassanello, 1921	51 M	Sigmoid flexure	Years	+	+	..	+	..	..	+	..	Resection	Recovered
Portis, 1922	40 F	Sigmoid flexure	Two months	+	..	+	+	+	+	+	+	.....	Passed spontaneously; recovered

TABLE I (*Continued*)

Author and Date	Age in Years and Sex	Situation of Lipoma	Duration of Symptoms	Pain	Constipation	Diarrhea	Blood in Stools	Loss of Weight	Anemia	Palpable Mass	Positive Roentgenologic Signs	Treatment	Remarks and Results
Derocque and Derocque, 1924	30 F	Cecum	Six years	+	..	..	..	..	..	..	..	Local excision	Recovered
Nash, 1924	61 F	Cecum	Six months	+	+	+	..	..	..	..	..	Local excision	Recovered
Dittrich, 1924[38]	..	Ascending colon	.....	+	+	..	..	..	..	..	..	Resection	Recovered
Vogeler, 1924	67 F	Rectum	Years	+	+	..	..	..	..	..	..	.....	Passed spontaneously; recovered
Partsch, 1924	59 M	Rectum	Three months	..	..	..	..	..	..	+	..	Local excision	Recovered
Belz, 1925–1926[11]	..	Ascending colon	.....	+	+	..	..	..	..	..	..	Resection	Recovered
Tanasescu and Barbilian, 1926	54 M	Transverse colon	Years	+	+	+	..	..	..	+	+	Resection	Recovered
Viannay, 1926	38 F	Sigmoid flexure	Four months	+	..	..	+	..	..	+	..	Local excision	Recovered
Douglas, 1926	41 F	Sigmoid flexure	.....	+	+	..	..	..	..	+	+	Resection	Recovered
Polák, 1928	47 F	Ascending colon	.....	+	..	..	..	..	..	+	..	Resection	Recovered
Matry, 1928	46 F	Cecum	Two weeks	+	+	..	..	..	..	+	+	Resection	Recovered
MacFarlane, 1928	47 F	Transverse colon	Nine months	+	..	..	+	..	..	..	+	Mikulicz's resection	Recovered
Pólya, 1929	45 F	Transverse colon	Eight months	+	+	..	+	+	..	+	+	Resection	Recovered
Mirolli, 1929	70 F	Probably transverse colon	One year	+	+	+	+	..	..	..	..	.....	Passed spontaneously; recovered
Stewart and Illick, 1930	40 F	Transverse colon	Nine years	+	+	..	..	+	+	..	+	.....	.....
Rouse and Mekie, 1930	47 F	Cecum	Three months	+	+	..	..	+	+	+	+	Local excision	Recovered
Ratcliff, 1930	57 M	Ascending colon	Three months	+	+	..	+	..	+	+	..	Local excision	Recovered
Ratcliff, 1930	60 F	Cecum	Two years	+	..	+	..	+	..	..	..	Resection	Recovered
Comfort, 1931	54 M	Sigmoid flexure	Six years	+	+	+	+	..	..	+	+	Resection	Recovered
Comfort, 1931	39 F	Ascending colon	One year	+	+	..	..	+	..	+	+	Colostomy and resection	Recovered
Comfort, 1931	43 F	Ascending colon	Eight years	+	+	+	..	..	+	+	+	Resection	Recovered
Webb, 1932	34 F	Cecum	Ten years	+	+	..	+	..	+	+	..	Resection	Recovered
Dull, 1932	63 F	Cecum	Five years	+	+	..	..	..	..	+	..	Enterectomy	Recovered
Brooks and others, 1933	54 M	Descending colon and sigmoid flexure	Three months	+	+	..	..	..	..	+	..	Local excision	Recovered
Lockhart-Mummery, 1933	50 M	.....	.....	+	+	+	+	..	..	..	+	Cecostomy and resection	Recovered
De Luca and Henstell, 1933	45 F	Cecum	Four months	+	+	..	..	..	+	+	+	Resection	Recovered
Panzner and Porretta, 1933	55 F	Transverse colon	Four months	+	+	..	..	+	..	+	+	Appendicostomy and resection	Recovered
Gordon, 1935	53 F	Cecum	One year	+	+	..	..	+	..	+	+	Resection	Recovered

between episodes. There is no apparent periodicity of the attacks, but they seem to come on at any time. The attacks are characterized by symptoms of mild intestinal obstruction, with general abdominal distress, distention, nausea, and often vomiting. The pain is usually colicky in nature and the attack is often terminated by a bowel movement. The relationship of the attacks to intake of bulky foods has been noted by several authors. These episodes may be the result of mechanical obstruction produced by the tumor itself or may be caused by intussusception of the intestine. It is probable that at first, when the tumor is small, the bouts of pain are caused by the tension of the tumor on the intestinal wall. As the tumor grows, the attacks become more frequent, and an irreducible intussusception may result.

TABLE II
CASES IN WHICH SUBMUCOUS LIPOMAS OF THE COLON DID NOT PRODUCE SYMPTOMS

Author and Date	Age in Years and Sex	Situation
Meckel, 1818[51]	F	Colon
Huss, 1844[51]	..	Sigmoid flexure
Wallis, 1884[51]	F	Colon
Hemmeter, 1901[51]	..	Colon
Kaufmann, 1922[14]	72 F	Transverse colon
Kaufmann, 1922[14]	62 F	Ascending colon
Comfort, 1931	48 F	Cecum
Comfort, 1931	63 F	Cecum
Comfort, 1931	63 M	Cecum
Comfort, 1931	35 M	Cecum
Comfort, 1931	65 M	Ascending colon
Comfort, 1931	65 M	Ascending colon
Comfort, 1931	62 F	Ascending colon
Comfort, 1931	65 F	Ascending colon
Comfort, 1931	70 F	Cecum and ascending colon
Comfort, 1931	51 M	Sigmoid flexure
Comfort, 1931	81 M	Rectum
Kirshbaum, 1935	60 F	Colon
Kirshbaum, 1935	83 F	Colon

In the second group of cases the symptoms are of short duration, and have been present only a few days or weeks. The patient has been in excellent health, and the attack begins very suddenly with sharp, cramp-like abdominal pain, which usually is situated at the site of the tumor. Nausea, vomiting and abdominal distention are present. The picture of acute intestinal obstruction is presented.

Pain was noted in 75 cases. This usually is the symptom that causes the patient to seek medical relief. It usually is colicky in character, and is sharp and brief. Occasionally, the pain is characterized by abdominal distress, which is vague in character and situation. The situation of the pain is sometimes over the site of the tumor, but when an intussusception is present, the pain may be far distant from the lipoma.

Constipation was noted in 49 cases; this was the chief intestinal irregularity in these cases. Many patients were relieved of their pain by taking a laxative and obtaining a bowel movement. Constipation alternating with diarrhea was noted in 13 cases, and diarrhea was noted in 7 cases.

Blood in or on the stools was noted in 27 cases. This apparently depends on ulceration of the overlying mucosa. The histories are not reliable in regard to blood because of the fact that the ordinary patient does not notice it if it is present, unless it is grossly evident. Chemical tests for blood are the only reliable criterion as regards its presence or absence.

Anemia was noted in 7 cases; in 3 of these patients the lipoma was in the cecum, and in 2 it was in the ascending colon. The exact reason for the anemia is not known, as is true concerning the profound anemia that often accompanies carcinoma of the cecum.

Loss of weight was noted in 15 cases. In some of these cases it was attributable to lack of eating for fear of bringing on an attack. Some patients learned that bulky foods would precipitate an attack, and, consequently, they went for weeks or even months, taking nothing but liquids by mouth. The majority of the patients, however, appeared well nourished and were usually overweight and not underweight.

In 64 cases it was possible to palpate a tumor either rectally or abdominally. In some of the cases in which the patient was seen early in the course of the disease, the tumor projected from the rectum. In the 3 cases which we have reported no masses were palpable. Case 3 in Comfort's series is of interest; here a mass was present at one examination, only to disappear and then reappear at the next examination. This was undoubtedly the result of a reducible intussusception.

A preoperative diagnosis of lipoma of the colon will seldom be made. Two more common conditions usually will be diagnosed, or the tumor may be considered to be a polyp.

Carcinoma of the colon will usually be the diagnosis in those cases in which the symptoms have been present for a long time. However, the history in cases of lipoma is usually one of periodic attacks with good health in between the attacks. Malignancy is associated with a progressive failure of general health. Patients who have carcinoma of the colon will seek medical aid earlier than will those who have a lipoma of the colon. Rankin, Bargen and Buie estimated that the average duration of symptoms of carcinoma of the colon is fifteen months, while in carcinoma of the rectum it is 11.7 months. In this series of cases, the duration of symptoms was noted specifically in 63 cases; the average duration was 41.5 months. In other cases in which the duration of symptoms was mentioned, the time was stated as "years"; probably, then, the average duration of symptoms was more than 41.5 months. Several authors have suggested that the presence of other fat deposits in the body should lead one to a diagnosis of lipoma. This is partly true because the patients usually appear healthy and overweight, but there are cases in which loss of weight has been marked and rapid, and in which anemia also has been present. The age at which lipomas chiefly occur is the age at

TABLE III

AGE AND SEX OF PATIENTS IN CASES OF SUBMUCOUS LIPOMA OF COLON AND RECTUM

Age in Years	Sex						Number of Patients
	In Cases in Which Symptoms Were Present			In Cases in Which Symptoms Were Absent			
	Not Given	Male	Female	Not Given	Male	Female	
Less than 20	0	0	1	0	0	0	1
20 to 29	0	1	4	0	0	0	5
30 to 39	0	3	10	0	1	0	14
40 to 49	1	10	17	0	0	1	29
50 to 59	0	18	10	0	1	0	29
60 to 69	0	1	7	0	3	5	16
70 to 79	0	1	1	0	0	2	4
80 to 89	0	0	1	0	1	1	3
Not given	10	1	0	2	0	2	15
Total	11	35	51	2	6	11	116

which cancer occurs, so this feature is of no value in differential diagnosis.

The roentgenologic studies of the colon by means of a barium enema may enable one to distinguish a benign from a malignant lesion. Weber has said:

> Lipomas of the large intestine are manifested by roentgenologic signs similar in all respects to those of other histologic types of sessile or pedunculated tumors. Typically they do not produce a genuine deformity of the peripheral contours of the shadow of the lumen distended with contrast material, but the defect they cause is a centrally placed, well-circumscribed, rounded or ovoid one within the lumen, and it is best elicited at the roentgenoscopic observation of the opaque enema.
>
> Ordinarily it is not possible to distinguish between the various histologic types of polypoid neoplasm. It is known, however, that adipose tissue, whether of neoplastic origin or not, is the most radiable of all the bodily tissues. Lipomas of the soft parts, especially of the extremities, are readily demonstrable roentgenographically as well-circumscribed shadows of diminished density surrounded by and in strong contrast to the adjacent much less radiable structures.
>
> Accordingly it has been possible with the use of the so-called double contrast method in the roentgenologic examination of the colon to suspect the true histologic nature of some of the lipomas encountered, simply by noting their failure to manifest the primary density possessed by intraluminal tumors of other histologic types, when they have become as large as lipomas ordinarily are when encountered. To be able to offer a diagnosis of this kind, however, it is essential that the technique of the roentgenologic examination be developed to an especially high degree of perfection.

If the tumor can be viewed through the proctoscope, the soft lobulated consistency and the yellow tinge of the mucosa overlying the tumor may lead to the correct diagnosis, but changes secondary to circulatory disturbances may alter the appearance of the lesion.

When the symptoms are of short duration the condition is difficult to distinguish from acute appendicitis, especially in cases in which the lipoma involves the cecum and ascending colon. However, the history of appendicitis is different; the attack begins with epigastric pain, which shortly is followed by nausea and vomiting, and localization of pain in the right lower abdominal quadrant, over the appendix. In appendicitis the pain is a constant aching and the patient tends to lie quietly. In obstruction caused by a lipoma the pain is intermittent and colicky, and the patient will often move about or rub his abdomen for relief. In obstruction, hyperperistalsis may be detected by sight or by auscultation. Palpation of a mass and abdominal distention are more marked in obstruction, while tenderness and rigidity characterize acute appendicitis. The most that can be done in cases of lipoma is to make a diagnosis of intestinal obstruction, but there is no reason to go further as to the etiologic agent of the obstruction. The general appearance of the patient and his former good health may lead one to make a diagnosis of a benign tumor.

The situation of the tumor is tabulated in Table IV. The chief sites of origin are, as Comfort found, in the cecum, ascending

Table IV
Situation of Submucous Lipomas of Colon and Rectum

Situation of Tumor	Cases		Total
	Symptoms Present	Symptoms Absent	
Ileocecal valve	1	..	1
Cecum	22	4	26
Ascending colon	16	5	21
Cecum and ascending colon	1	1	2
Transverse colon	13	1	14
Probably in transverse colon	1	..	1
Descending colon	9	..	9
Sigmoid flexure	12	2	14
Descending colon and sigmoid flexure	2	..	2
Rectum	11	1	12
Colon	5	5	10
Probably in colon	4	..	4
Total	97	19	116

colon and sigmoid flexure, in the order named. The tumor involved the right half of the colon in 50 cases, the transverse colon in 15 cases, and the left half of the colon, including the rectum, in 37 cases.

Comfort has described very thoroughly the pathologic anatomy of these tumors. The mucosa overlying the tumor may be atrophic or ulcerated or may be perfectly normal in appearance. The muscularis mucosae is very faint in the large tumors, but can be traced intact over the small ones.

The thinning of the muscular layer of the intestine in the large tumors causes a peculiar yellow coloration on the external surfaces, which is the result of the fat. This led several observers to make the diagnosis at operation.

The shape of the tumor varied with the type of attachment and the size of the tumor. If attached by a fine pedicle, the tumor may be oval, flattened or elongated, whereas, if the attachment is by a broad base, the tumor varies in shape from that of a disk to that of a pear. The surface may be rounded and smooth, or irregular and lobulated.

The fat cells are usually of the adult type. The line of demarcation between the submucosal connective tissue and the fat becomes more definite in the larger tumors and there often is a definite capsule surrounding the fat. Septums divide the tumors into small lobules. Arterioles and numerous capillaries with supporting stroma are present.

Interference with the circulation of these tumors leads to edema, congestion, ulceration, hemorrhage into the substance of the tumor, infiltration with leucocytes, and necrosis.

Although benign, these tumors may undergo necrosis, cystic formation, calcification, and myxomatous or sarcomatous degeneration.

TREATMENT

Submucous lipomas of the colon are benign growths and do not require the extensive dissection of lymph nodes that is necessary in cases of carcinoma. Local excision of the growth and direct anastomosis is an ideal procedure, but this should be undertaken only when the patient is an exceptionally good risk and on an unobstructed bowel. If obstruction should be marked, decompressive measures may be necessary before the resection is performed. In operations on the colon, it appears that operations in stages are preferable to extensive single stage resections. An ileocolostomy and resection at a second stage is a preferable procedure on the right half of the colon. For tumors situated in the transverse, descending or sigmoid colon, an operation in which the segment of the bowel containing the tumor is resected extraperitoneally is to be preferred, such as the Mikulicz operation or some modification of its principle. Local excision of lipomas of the rectum may be accomplished through the proctoscope without a great deal of risk.

There were 9 deaths among 83 cases in which a surgical procedure was employed. Most of these deaths occurred in the early years; the most recent death was in 1921. Thirty-three patients have been operated on since then without a death.

SUMMARY

A review of the literature revealed 113 cases of submucous lipoma of the colon and rectum; in 94 of these cases the tumor produced symptoms. Three cases in which the condition was treated successfully with operation have been added. Submucous lipomas of the colon and rectum chiefly affect women who are between forty and sixty years of age. The tumors are found in the cecum, ascending colon, and sigmoid flexure, in the order named. The condition rarely is diagnosed preoperatively; the most common diagnosis is carcinoma and acute appendicitis. The symptoms are those of intestinal obstruction, and the average duration of symptoms is 41.5 months. The treatment is surgical removal

in one stage, if possible, or by means of graded procedures.

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REVIEW OF ULCER SURGERY*

AT THE PRESBYTERIAN HOSPITAL 1921 TO MARCH 1935 INCLUSIVE

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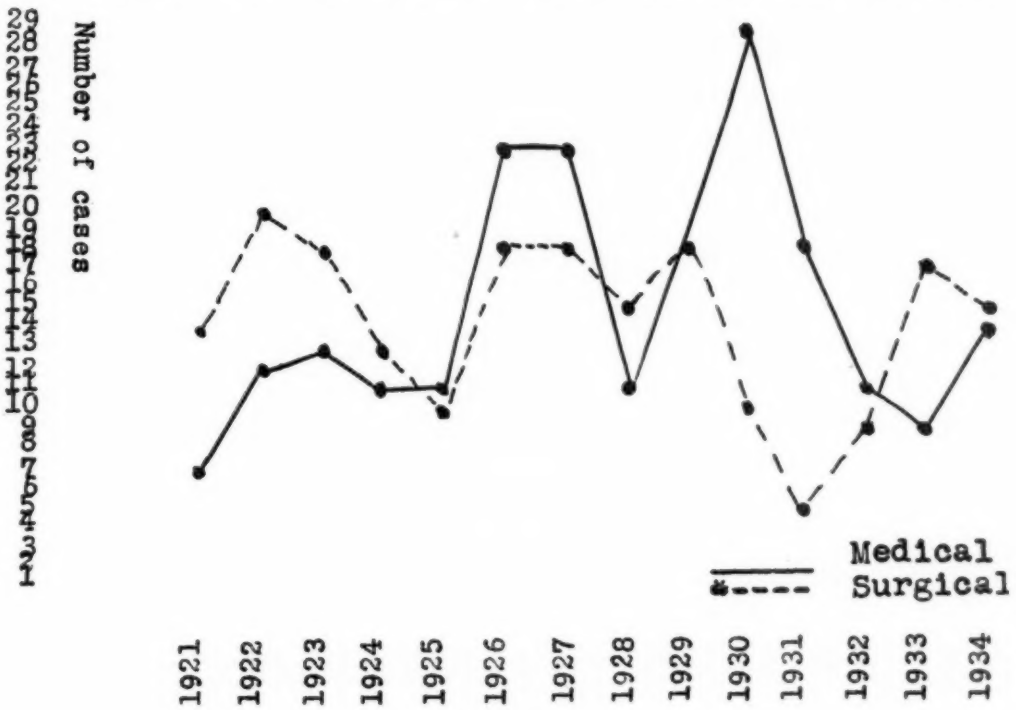
DURING this period 415 cases of peptic ulcer have been admitted, 209 medical and 206 surgical. In 1929 an equal number was admitted upon each service. During 1930 and 1931 the medical cases more than doubled the



GRAM 1.

The respective relationship of these two groups is illustrated in Gram 1 and Table 1.

These figures are actual, and denote true surgical and medical cases. A patient admitted to the Medical Ward and subsequently transferred for surgical intervention is considered solely as a surgical case. During the years 1921–1924 surgical cases greatly out numbered the medical cases, while in 1925, 1926 and 1927 they were more relatively equal. In 1928 there was a marked fall in the number of medical cases with a relative increase in the surgical. surgical admittances while in 1933 the reverse is true. It is interesting to note that in spite of the marked difference in actual admissions during the individual years, the total numbers for fourteen years are approximately equal.

Gram 2 denotes the relationship between the elective cases and the surgical emergencies, which together total the operative group.

The former consists of 125 elective surgical cases; 104 males and 21 females. Ages range from 7 to 70 years. Of the 107 actual ages given there are three between

* From the Department of Surgery, Presbyterian Hospital, Philadelphia, Pa.

TABLE I

	1921	1922	1923	1924	1925	1926	1927
Medical	7	12	13	11	11	23	23
Surgical	14	20	18	13	10	18	18
A	10	12	11	6	5	12	14
B	4	8	7	7	5	6	4

	1928	1929	1930	1931	1932	1933	1934
Medical	11	18	28	18	11	9	14
Surgical	15	18	10	5	9	17	15
A	10	14	5	4	5	8	8
B	5	4	5	1	4	9	7

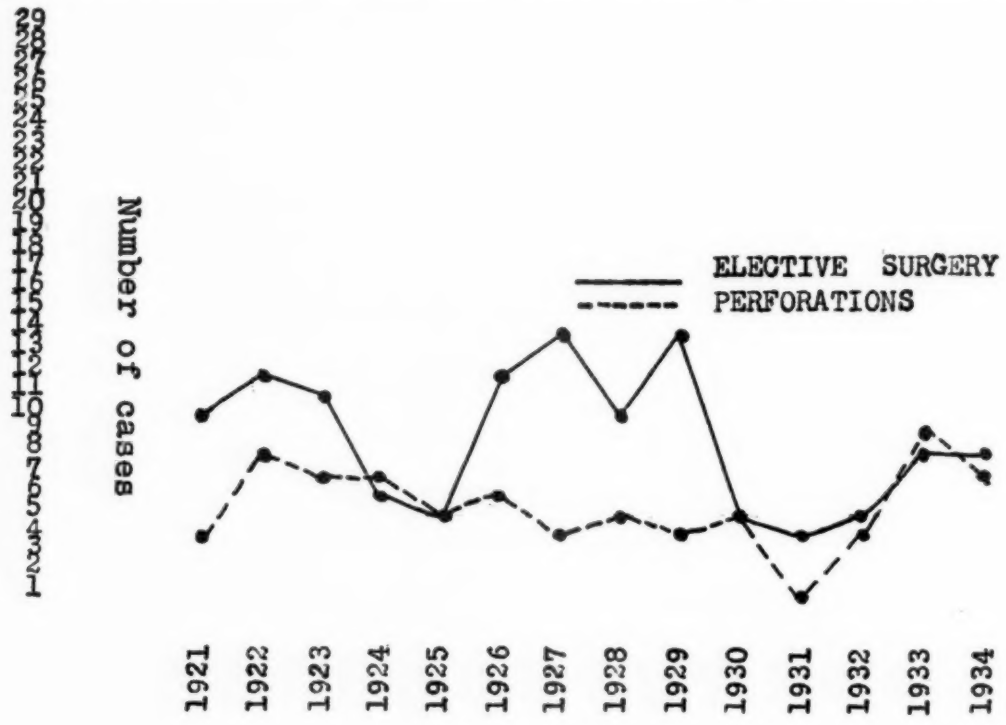
A—Elective surgery.
B—Perforations.
Total medical..... 209
Total surgical..... 206 (Perforations 81)

ages of 7 and twenty years, thirteen between 21 and 30 years, twenty-two between 31 and 40 years, thirty-four between 41 and 50 years, twenty-four between 51 and 60 years, and eleven between 61 and 70 years. Presenting a percentage of 23.36 during the second and third decades as compared with 54.2 per cent during the fourth and fifth decades.

In ninety-one of the considered cases, definite reference as to the duration of ulcer symptoms is given and denotes a variation from three months to thirty-five years. Specific age and symptom relationship are given in eighty cases. This is shown in Table II.

TABLE II

Age	No. of Cases	Average Duration of Symptoms
7	1	Since birth
16	1	Four months
19	1	Three years (Melena)
21–30	9	4.166 years
31–40	20	7.33 years
41–50	27	8.07 years
51–60	16	10.6 years
61–70	5	10.8 years
Total	80	



GRAM 2.

These figures denote a proportionate increase in symptom duration with each

decade; 58.6 per cent of these cases were operated upon during the third and fourth decade during which period the average duration of symptoms was 7.7 years. Total average duration was 6.31 years. Woolsey (1934) reports a symptom duration of 6.38 years.

TABLE III

Duodenal	Pylorus	Lesser Curvature	Greater Curvature	Anterior Wall	Posterior Wall	Lesser Curvature Near Cardia	Gastric Near Pylorus	Marginal	Total
78	21	13	1	1	5	2	3	1	125
62.4%	16.8%	10.4%	.8%	.8%	4%	1.6%	2.4%	.8%	100%

LOCATION OF ULCER

In two cases multiple gastric ulcers are recorded, and in one, an associated malignancy of the cecum and ascending colon. The remaining are specifically located as follows in Table III.

Definite pyloric stenosis was recorded in 9 duodenal cases and four pyloric, and in one case a pyloric ulcer was questionably malignant.

Operative endeavors have been practiced with a marked variance of procedures. Posterior gastroenterostomy with its various modifications and associations, has been the procedure of choice in 112 of the recorded cases (Table IV).

Eighty-four or 67.2 per cent of the operated cases recovered without complications and were discharged from the hospital in an average of 19.45 days postoperative. Twenty or 16 per cent suffered complications but recovered and were discharged at an average of thirty-two days postoperative, the remaining twenty-one cases or 16.8 per cent died. Total percentage of those suffering complication is 32.8 per cent.

Persistent emesis without obstruction and pulmonary involvement in the nature of laryngitis, bronchopneumonia, pleurisy, and pulmonary embolus account for 58.33 per cent of the complications suffered by the patients who recovered. Intestinal obstruction occurred in two instances; pyelitis, thrombophlebitis and parotitis in separate cases respectively; delayed healing of the wound was a complicating factor in two cases; and an atropine reaction in another.

TABLE IV

Surgical Procedure	No. of Cases
Posterior Gastroenterostomy	
Without allied surgical procedure	39
With simple cautery of ulcer	8
With appendectomy	44
With simple cautery and appendectomy	5
With appendectomy and cholecystectomy	6
With Balfour excision of ulcer	2
With cholecystectomy	1
With simple cautery and cholecystectomy	1
With an associated jejunostomy	1
With the Lahey modification of Polya operation	1
Billroth No. 2	4
Anterior gastro-enterostomy	2
Anterior gastro-enterostomy with enteranastomosis	2
Simple excision of ulcer and appendectomy	2
Simple cautery of ulcer and appendectomy	1
Horsely pyloroplasty	2
Pyloroplasty, Heineke, Michulitz with Judd modification	1
Finney pyloroplasty with associated repair of marginal ulcer	1
Ileostomy followed in 14 days with a posterior gastro-enterostomy	1
Ileostomy, partial gastrectomy with a Polya anastomosis	1
Total	125

	Average Stay in Hospital, Days
84 or 67.2% recovered without complications	19.45
20 or 16% suffered complications	32
21 or 16.8% died	

The mortality group consists of four females and seventeen males, the age ranging from 28 to 66 years, 38.1 per cent occurring between the ages of 41 and 50 and

47.6 per cent between the ages of 51 and 60 years. Fifteen of the deaths occurred within five days postoperative while the remaining six died within eight and forty days postoperative.

Mechanical obstruction was the cause of death in 5 cases; the operations in association were posterior gastroenterostomy, 1 case; posterior gastroenterostomy with appendectomy, 2 cases; partial gastric resection and posterior gastroenterostomy, 1 case; and posterior gastroenterostomy with cholecystectomy, 1 case.

Myocardial failure with terminal pulmonary edema occurred in 5 cases; pneumonia in 6 instances.

Pulmonary embolus occurred in one case following posterior gastroenterostomy and appendectomy. Hematemesis with inanition was the stated cause of death in one case following partial gastric resection and posterior gastroenterostomy.

Death followed within approximately forty-eight hours in 2 cases in which anterior gastroenterostomies were performed, and in one case following posterior gastroenterostomy no immediate cause of death was given.

According to the statistical reports of Luff, Balfour, Moynihan, Walton, Walters and Woolsey, gastroenterostomy is the operation of choice in the treatment of peptic ulcers, particularly duodenal which have not responded to medical therapy. It not only "alters the mechanics and chemistry of the stomach, but diminishes the material passing through the duodenum, reduces the emptying time of the stomach, relieves pyloric stenosis, lowers the acidity by drainage and neutralization and thus relieves pylorospasm," all results to be desired in a successful operation.

Luff reports satisfactory results in 89.5 per cent in a series of 2609 cases. Balfour in a recent article advocates local removal, by wedge shaped incision, cautery combined with gastroenterostomy or pyloroplasty for small ulcers and partial gastrectomy and reconstruction by Billroth I and II for large ulcers.

The actual mortality following gastroenterostomy in this series is 12.7 per cent. Woolsey reports a mortality of 2.7 per cent and Mayo sets the mortality of gastroenterostomy in all classes of ulcers, perforating, chronic and acute, at 2.85 per cent. Whether or not these figures represent both the surgical mortality and deaths resulting from chest complications, etc., we have not been able to ascertain.

Four of the 5 cases in which partial gastrectomy was performed died.

Of the acute surgical emergencies, first seen in the Accident Ward of this institution, perforated peptic ulcer plays a most significant role. During the period in question eighty-two such cases have been admitted, seventy-two acute perforations, seven subacute perforations, and three questionable perforations (patients admitted in shock and clinical diagnosis not confirmed either by surgical intervention or postmortem examination).

Four cases of recurrent perforations are included. No cases of multiple perforations are recorded.

Four female patients, ages 50, 51, 69 and 71 years suffered perforations in the cardiac end of stomach, lesser curvature of stomach, jejunal margin, and pylorus, respectively. In one case the perforation was discovered postmortem. In the remaining three, operation took place between nineteen and seventy-two hours following the acute onset. Three patients subsequently died of generalized peritonitis, the other from cardiac failure. This series represents a rather high incidence of perforations occurring in female patients, namely 1 to 18 as compared with Eliason, 1 to 31 cases (series of 2630), Stewart and Barber and Patterson record an incidence of 1 to 5 (series of 36 cases). The prevalence of gastric perforation is in keeping with cases recorded by Enoch and Dansey.

PREVIOUS ULCER HISTORY

Previously considered and diagnosed ulcer history is given in forty-five cases of

the acute perforations, twelve of which were receiving medical treatment at the time of perforation. Duration of symptoms varied from one to twenty-five years. Fifteen cases presented typical ulcer histories ranging from five days to two years, but were undiagnosed until the time of the surgical emergency. Eight cases or 11 per cent denied previous ulcer history. Eliason reports an incidence of about 18 per cent.

Deaver reports a 90 per cent incidence of previous ulcer history as compared with 83.33 per cent in this series. Three presented an atypical picture.

The seasonal time of perforation has been well distributed throughout the year. In 23 or 36.5 per cent of the cases in question, no exciting or attributing factor associated with the acute onset is mentioned. In 11 cases perforations occurred while patients were at physical labor, 3 followed a heavy meal, 5 followed alcoholic debauch, 1 associated with bowel movement, 1 following intake of ice water, 3 after taking a cathartic and 1 as a sequel to a gastrointestinal study. In four cases patients were awakened with the acute onset and in sixteen cases the onset followed as an acute exacerbation of a previously existing abdominal pain of varying duration. Three cases were admitted in a collapsed condition and diagnosis was confirmed postmortem.

Symptoms and signs are considered in order of their occurrence and significance.

Pain typified by such terms as acute, sharp, violent, agonizing, intense, griping, excruciating, cutting, prostating and unendurable is the initial complaint in every case.

The stated location was abdominal in 39.4 per cent of cases, epigastric in 26.7 per cent, upper abdominal in 18.3 per cent, lower abdominal in 5.6 per cent, and generalized abdominal in seven of the remaining 7 per cent of cases.

Pain reference is recorded in 14.5 per cent of cases: to the back in one case, to back and right shoulder in two instances, to right shoulder alone in five cases, to both shoulders upon two occasions, and to the left shoulder alone in one instance. In 1 case the pain radiated from the epigastrium to the scrotum. The pain once established was constant in the majority of cases in which this fact was recorded, and intermittent in several instances; a definite period of quiescence is recorded in three cases.

The immediate effect of the pain in ten instances caused the patient to double up. Collapse, total or partial, weakness, faintness occurred in approximately 25 per cent of the cases recorded; profuse perspiration and pronounced thirst, anxious expression and extreme pallor characterized these conditions in many instances. No blood pressure readings were taken.

Nausea without vomiting occurred in eight cases and was associated with vomiting in five cases. Vomiting was recorded in thirty-three cases or 46.5 per cent. Hematemesis was noted in 4 instances and melena in one.

Tenderness (Table v) was recorded in forty-five of sixty-three cases of acute perforation. Specific location was as follows: generalized in 31 cases, 20 duodenal, 7 gastric and 4 pyloric; both upper quadrants in 4 cases, 3 duodenal and 1 pyloric; right upper and lower quadrants in 6 cases, 4 duodenal and 2 gastric; epigastrium in 3 cases, 2 duodenal and 1 pyloric; right upper quadrant, 1 case of duodenal perforation.

Board-like rigidity (Fig. 5) was recorded in sixty-three cases; it was generalized in 39 cases, 25 duodenal, 7 pyloric and 7 gastric perforations; localized, to both upper quadrants in 5 cases, 4 duodenal and 1 pyloric; to right upper and lower quadrants in 2 cases, 1 gastric and 1 duodenal; to right upper quadrant alone in 5 cases, 3 duodenal and 2 gastric and in the right lower quadrant in one instance of duodenal perforation.

Spasticity and mild rigidity occurred in 11 cases, 7 duodenal and 4 gastric. Seven of these cases were first seen more than six hours after the acute onset and four

TABLE V

PHYSICAL SIGNS IN OPERATED CASES

	Within 6 Hours			6 to 12 Hours			12 to 24 Hours			Over 24 Hours			Total Number of Cases
	G	P	D	G	P	D	G	P	D	G	P	D	
TENDERNESS													
Generalized tenderness	4	3	10	..	..	6	2	1	4	1	..	..	31
Tenderness most marked in both upper quadrants	..	1	1	..	..	1	..	..	..	..	..	1	4
Right upper and lower quadrants	1	..	1	..	..	2	..	..	1	1	..	..	6
Epigastrium	..	1	1	..	..	1	..	..	..	..	..	..	3
Right upper quadrant	..	..	1	..	..	..	..	..	..	..	..	..	1
Not recorded in	1	..	9	..	1	3	1	..	2	1	..	..	18
RIGIDITY													
Generalized (boardlike)	3	5	14	..	1	7	3	1	4	1	..	..	39
Both upper quadrants	..	1	2	..	..	1	..	..	..	..	..	1	5
Right upper and lower quadrants	1	..	1	..	..	..	..	..	..	..	..	..	2
Right upper quadrant	2	..	1	..	..	2	..	..	..	..	..	..	5
Right lower quadrant	..	..	..	..	..	1	..	..	..	..	..	..	1
Spasticity and mild rigidity	2	..	2	..	..	2	..	..	3	2	..	..	11
Not recorded in													
Peristalsis absent in	2	1	6	..	..	4	3	1	2	3	..	..	22
Peristalsis diminished in	2	2	1	..	..	2	..	..	..	..	..	..	7
Peristalsis present in	..	..	..	..	..	1	..	..	1	..	..	..	2
Not recorded in	2	..	18	..	1	6	..	..	4	..	..	1	32
Peritoneal tenderness	..	..	..	..	..	1	..	..	1	..	..	..	2
Dullness in flanks	1	..	..	..	..	..	2	..	..	..	..	..	3

TABLE VI

Cases Operated	Cases*	Temperature, Degrees F.			Pulse			Respirations			Cases*	White Blood Count		
		Low	High	Average	Low	High	Average	Low	High	Average		Low	High	Average
Within 6 hours	35	98	100	97.4	64	108	88	16	36	26	19	7,800	19,500	14,850
Between 6 and 12 hours	14	97	101	98.3	72	120	93	18	40	24	10	8,700	21,650	14,700
Between 12 and 24 hours	11	95	101	97.1	72	136	98	24	40	28	4	8,100	17,500	14,325
Over 24 hours	5	99	100	99.1	86	124	100	18	30	23	5	5,100	29,600	18,070
Total	65	..	...	....	..	...	...	..	..	..	38			

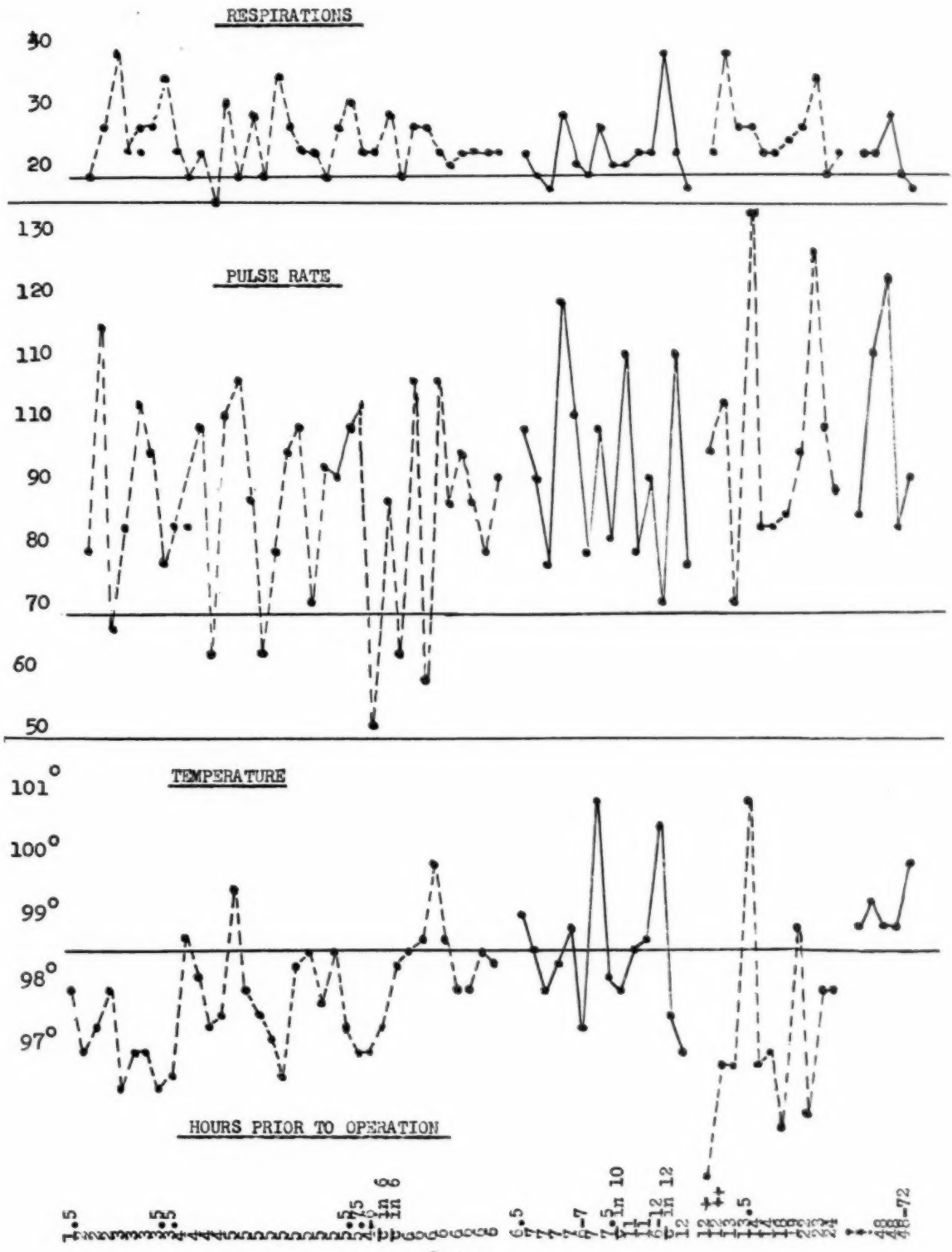
* No. of cases in which these points were recorded.

before the six hour limit. Distention was invariably associated.

Peristalsis was absent in twenty-two, diminished in seven and active in two in thirty-one cases in which this point was recorded.

Peritoneal tenderness was elicited upon two occasions and dullness in flanks upon percussion was ascertained in 3 instances.

In over 95 per cent of the cases operated upon within six hours after the acute onset, the temperature (Table VI and Gram 3) is either normal or subnormal, with an associated increase in pulse and respiration. The average for 35 cases in this series in


RESPIRATIONS
40
30
20
PULSE RATE
130
120
110
110
90
80
70
60
50
TEMPERATURE
101°
100°
99°
98°
97°
HOURS PRIOR TO OPERATION

Gram 3.

which these points are recorded is temperature 97.4, pulse rate 88, and respirations 26. Cases operated upon between six and twelve hours have a slight elevation of temperature and pulse rate, as compared with those operated upon within the six hour limit with little change in the respiratory rate. The average for 14 cases in this series is temperature 98.3, pulse rate 93, and respirations 24. Cases operated upon between twelve and twenty-four hours with few exceptions have a subnormal temperature with an increase in pulse rate; the respiratory rate showing a slight increase. The average for 11 cases is temperature 97.1, pulse rate 98, and respirations 28.

In cases operated upon over twenty-four hours after the acute onset, the temperature and pulse rate invariably increase with little change in the respiratory rate. Five cases, so operated upon in this group, have an average temperature rate of 99.1°, pulse rate 100 and respirations 23.

The leucocytic response to such a calamity shows a greater proportionate increase from the normal in those cases operated upon within the six hour limit, as compared with those operated upon within the twelve and twenty-four hour limit, respectively. In those cases operated upon over twenty-four hours the leucocytic count shows a considerable increase. In all, 38 white blood counts were taken in this series (Table VI).

ANESTHESIA

Nitrous oxide, oxygen and drop ether were the anesthetics used in 77.5 per cent of the acute perforations. Spinal was used in 4 instances, ether alone in 3 cases, nitrous oxide and oxygen alone in 3 cases, local in 3 cases, and local supplemented by gas in one instance.

The duration of anesthesia varied from twenty-five minutes to one hour and fifty minutes. The average duration in cases which suffered no complications was approximately sixty minutes as compared with sixty-eight minutes in those which suffered chest complications.

In all of the operated cases, 66 in number, free fluid, gastric contents, air or pus were in the abdominal cavity.

Table VII and Figure 1 illustrate the location of 201 peptic ulcers which include both the elective and emergency group. In addition there were 3 marginal ulcers, one of which was an elective case and the other 2 were surgical emergencies. In the elective group the ratio of gastric, including pyloric, to duodenal ulcers was 1 to 1.6, while in the emergency cases the ratio was 1 to 1.7.

The actual percentage location of this ulcer group is as follows: duodenal 62.75, junction of duodenum and pylorus .49, pyloric 14.7, gastric 20.58 and marginal 1.5.

Rivers of the Mayo Clinic, reports a ratio of 9 duodenal to 1 gastric ulcer in a series of 2499 cases. Brown reports ratio 3.24 duodenal to 1 gastric in a series of 695 cases. Shawan reports an incident ratio of 1 gastric to 1.56 duodenal in series of 159 perforations.

Table VIII illustrates the type of operation performed in relation to the location of the perforation and the time of operation.

TABLE VII

LOCATION OF ULCER (PERFORATED)

Duodenum	Junction of Duodenum and Pylorus	Pylorus	Marginal	Stomach					Total
				Cardia	Anterior Wall	Posterior Wall	Lesser Curvature	Greater Curvature	
50	1	9	2	1	3	2	11	0	79

OPERATIVE PROCEDURE

Thirty-two, or approximately 49.2 per cent of these perforations, were closed either by simple closure, cautery or inversion, and the abdomen closed with drainage. In 6 instances the abdomen was closed without drainage. Posterior gastroenterostomy was added to the above procedure in fourteen cases, ten of which were drained and 4 were not. The appendix was removed in six instances, one in conjunction with simple closure and posterior gastroenterostomy, four with simple closure of the ulcer, and in one a pyloroplasty was performed. Jejunostomy was performed in 2 cases, both in conjunction with simple closure. Pyloroplasties, Heineke-Mikulicz with Judd modification were performed upon two occasions. In 1 case the duodenum was sutured to the undersurface of the liver. Cautery followed by a Cushing repair and gastroenterostomy was performed in 1 case and in 2 instances perforations of marginal ulcers were repaired. In one case of which the original gastroenterostomy was separated, the stomach opening closed, a portion of the jejunum resected and an end-to-end anastomosis performed. In the other case the ulcerated area was cauterized and the adjacent edges approximated by three rows of sutures.

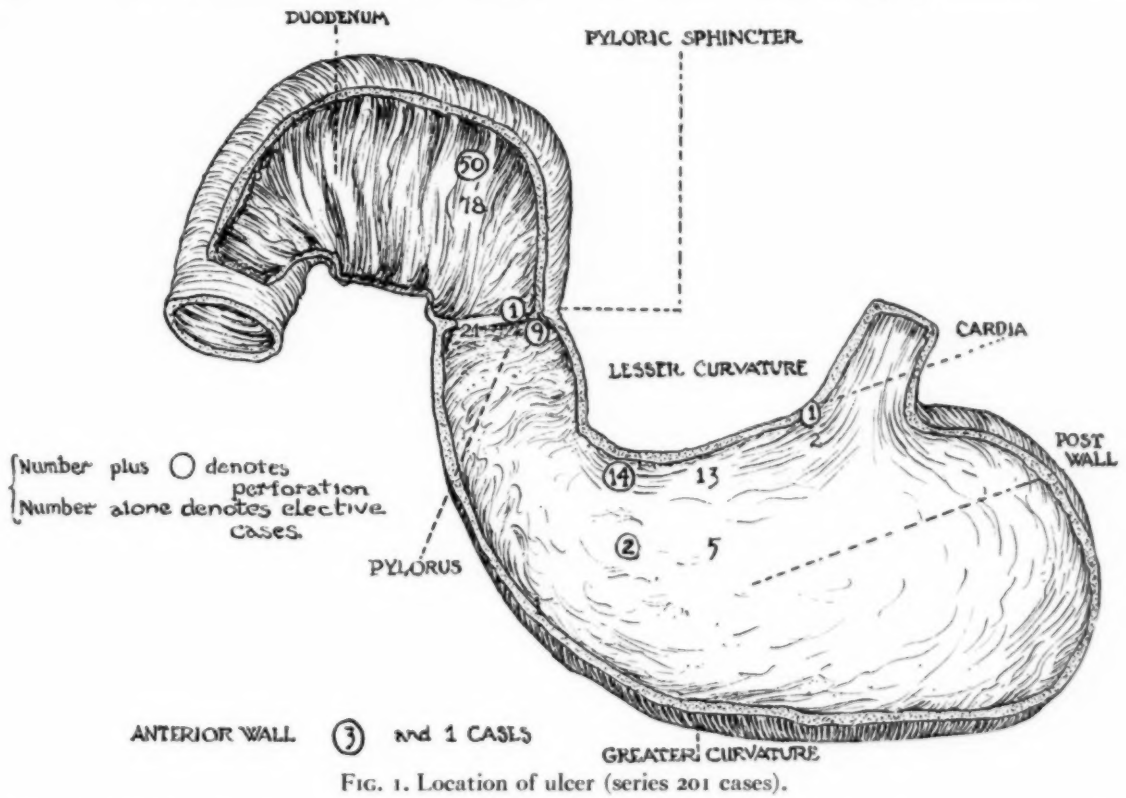


FIG. 1. Location of ulcer (series 201 cases).

In this series the abdomen was drained in 68.5 per cent of the cases operated upon within six hours, in 85.7 per cent of cases operated upon between six and twelve hours, and in 100 per cent of the cases operated upon over twelve hours following the acute onset. Trout concludes from the report of 100 surgeons that 80 per cent as a rule do not use drainage, 20 per cent drain in every case, 60 per cent drain in late cases, and 20 per cent never drain under any circumstance.

COMPLICATIONS WITH RECOVERY

Twenty-one of the 35 cases of acute perforations operated upon within the six hour limit suffered no postoperative complications; the average duration in the hospital was twenty days.

TABLE VIII

Operative Procedure	With Drainage	Without Drainage	Gastric				Location of Perforation								Total
							Pyloric				Duodenal				
			Operation Occurring												
			Within 6 Hrs.	6–12 Hrs.	12–24 Hrs.	Over 24 Hrs.	Within 6 Hrs.	6–12 Hrs.	12–24 Hrs.	Over 24 Hrs.	Within 6 Hrs.	6–12 Hrs.	12–24 Hrs.	Over 24 Hrs.	
Simple closure	17	3	1	..	2	2	2	1	1	..	7	3	1	..	20
Cautery and simple closure	6	1	2	..	..	..	1	..	..	..	1	2	1	..	7
Cautery and inversion	3	..	..	..	1	..	..	..	..	..	..	..	2	..	3
Simple cl. and post. gast	4	1	1	..	..	..	..	..	..	..	2	2	..	..	5
Inversion	6	2	2	..	..	..	2	..	..	..	3	1	..	..	8
Inversion and post. gast	1	..	..	..	..	..	1	..	..	..	..	..	..	..	1
Cautery inv. and post. gast	1	1	..	..	..	..	..	..	..	..	2	..	..	..	2
Cautery simp. c. and post. gast	4	2	..	..	..	..	..	..	..	..	4	1	1	..	6
S. clos. post. G. and Append	..	1	..	..	..	..	..	..	..	..	1	..	..	..	1
Caut. simp. c. and append	1	..	..	..	..	..	..	..	..	..	1	..	..	..	1
Pyloroplasty (H. M. and J) append.	1	1	..	..	..	..	..	..	..	..	1	..	1	..	2
Simple closure and append	2	..	..	..	..	..	..	..	..	..	..	1	..	1	2
Simp. clos. and jejunostomy	2	..	..	..	..	1	..	..	..	..	..	1	..	..	2
Cautery inv. post. g. and append	1	..	..	..	..	..	..	..	..	..	..	1	..	..	1
Cautery (Cushing R) and post G	1	..	..	..	..	..	..	..	..	..	..	1	..	..	1
Duodenum sut. und. s. of liver	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1
Marginal repair	1	1	..	..	..	..	..	..	..	..	..	..	..	..	2
	52	13	..	..	..	..	..	..	..	..	..	..	..	..	65

In 12 of the remaining cases, intestinal obstruction occurred in 2 instances, partial evisceration in one case, complete evisceration in another, and in 2 cases there were wound infections. Pneumonia occurred in one instance. The average duration in the hospital was thirty-two days.

Four of the fourteen cases operated upon between six and twelve hours suffered no complications and were discharged from the hospital at an average of sixteen days.

Pneumonia with recovery occurred in 3 instances, atelectasis and incisional abscess in 1 case, and pyloric obstruction in another. Average duration in hospital of this group of five cases was thirty-eight days.

Two of the 11 cases operated upon between twelve and twenty-four hours recovered, one in which a simple closure and posterior gastroenterostomy was performed suffered no complications and was discharged twenty-seven days postoperative, the other in which a simple closure was done had a slight wound infection but was also discharged in twenty-seven days. No cases operated upon after the twenty-four hour limit recovered.

MORTALITY

Six proved cases of perforation, 4 duodenal, 1 pyloric, and 1 cardiac, were admitted in shock and died without operation shortly after admission.

Death occurred four days postoperative in a case of a male, aged forty-four years, operated upon two hours after acute onset, simple duodenal closure posterior gastroenterostomy and appendectomy had been performed; the immediate cause of death was lobar pneumonia and pulmonary edema.

Subdiaphragmatic abscess with associated pleural effusion was the immediate cause of death thirty-three days postoperative in the case of a male, aged twenty years, operated upon five hours after the acute duodenal perforation. A simple closure with drainage had been performed.

Generalized peritonitis was the cause of death three to five days postoperative in 3 cases of duodenal perforation operated upon between six and twelve hours.

Bronchopneumonia followed simple closure of a pyloric ulcer in one case, death occurring sixteen days postoperative. In one case following duodenal perforation auricular fibrillation with terminal pulmonary edema occurred four days postoperative.

Death occurred following 2 gastric, 1 pyloric and 6 duodenal perforations in cases operated upon between twelve and twenty-four hours. Generalized peritonitis was the cause in 4 instances, cardiac failure in 3, cardiac failure generalized peritonitis in 1 and intestinal obstruction and generalized peritonitis in another. In this group 6 simple closures with drainage were performed, 1 simple closure drainage and appendectomy, 1 pyloroplasty, and in one instance the duodenum was sutured to the undersurface of the liver.

Likewise, peritonitis was the chief complicating factor in cases operated upon over twenty-four hours. It occurred in 4 instances, in 1 of which there was an associated parotiditis and lobar pneumonia. Complete evisceration with associated acute gastric hemorrhage was the cause of death in one case. This group includes 3 gastric, 1 marginal and 1 duodenal perforation. Simple closure was performed in 3 instances, simple closure and jejunostomy in one and simple closure and appendectomy in one. All cases were drained.

The respective mortality in these four groups are 5.7 per cent within six hours, 35.7 per cent in six to twelve hours, 81.8 per cent in twelve to twenty-four hours, and 100 per cent over twenty-four hours. The average operative mortality was 31.8 per cent.

The total mortality was 37.5 per cent. Butler records a mortality rate of 24.51 per cent Eliason 47.2 per cent (74 cases); Meyers 29 per cent (62 cases).

From this review a rather clear conception of the clinical picture of an acute surgical emergency may be readily observed. In acute peptic ulcer perforations, males outnumber females in a ratio of 18 to 1, the age incidence may vary from 19 to 71, with over 71 per cent occurring between the third and sixth decades. A previous history of ulcer disturbance may be obtained in the majority of cases, although due to the acuteness of the attack this fact may not be ascertained until many hours postoperative. The onset is acute and may be associated with alcoholic indiscretions, excessive intake of food, strenuous exercise or as a sudden exacerbation of preexisting abdominal distress. In many cases, however, no associated factor is related and the patient may be suddenly taken with an acute abdominal distress which is primarily in the nature of a sudden excruciating pain, causing the patient to be "struck motionless" or doubled up.

The abdominal pain may be definitely localized either in the epigastrium, upper or lower abdomen. It may be constant or intermittent, and in a small percentage of cases may tend to radiate by way of the costal margins to the back, interscapular region and right and left shoulders, respectively.

The general appearance of the patient is frequently quite characteristic; anxious fixed expression, pallor, profuse perspiration and associated polydypsia. The position of the abdomen is fixed and the thighs are invariably flexed upon the abdomen. Patient may be lying perfectly still or may be squirming with pain.

Distention may or may not be present. Rigidity (board-like in nature) is a most constant sign and may be generalized or localized in the epigastrium, upper right and left quadrants, right upper and lower

quadrants, and occasionally in the right upper quadrant alone, associated with which is extreme tenderness, which is so intense in some cases that the patient may forbid palpation.

Occasionally the abdomen may be soft and the tenderness less marked. Such was the case in one of the more recent perforations seen in the accident ward. The patient, a man aged fifty-three years, walked into the receiving ward complaining of severe abdominal pain of one-half hour duration, which at the onset caused him to faint and his abdomen to become hard. He denied any alimentary indiscretion, but stated that three years previously, following an *x*-ray study, he had been put upon an ulcer regime, to which he had remained faithful until one month previous to present onset. He was placed upon the examining table and his abdomen examined. It was very soft but very tender particularly in the gall-bladder region. Peristalsis was somewhat diminished and he complained of nausea. His temperature was subnormal, pulse rate and respirations slightly increased. One of the attending men, not knowing the previous history of the man, regarded the case as one of acute cholecystitis. The surgeon had scarcely left the examining room when the patient was again suddenly seized with an excruciating abdominal pain which doubled him up and radiated to the back and shoulder regions. His expression became anxious, his skin moist and cold, he perspired profusely, complained of intense thirst, and in a period of several seconds his abdomen became board-like. He was immediately admitted and a perforated gastric ulcer was revealed at operation. A period of quiescence had evidently intervened between the two attacks.

Peristalsis is usually absent or diminished. Nausea and vomiting are not infrequent symptoms, but their absence or presence offers but few points of differentiation. The presence of liver dullness was not ascertained in any of the cases recorded, peritoneal tenderness was elicited upon the two occasions such examinations were attempted; likewise dullness in flanks was recorded in three instances.

The first temperature taken is usually subnormal varying in degree to the duration of the acute onset while the pulse rate and respirations are invariably increased.

The leucocytes show an active response during the first six hours following perforation with little change in the subsequent twelve to eighteen hours. The urine shows no characteristic features.

SUMMARY

Several paramount facts have been well impressed upon us; primarily, the importance of adequately informing all known ulcer cases as to the seriousness of their condition and the possibility of their developing an acute surgical emergency at any time.

Secondly, the significance of being well acquainted with the signs and symptoms of the acute abdomen; a correct early diagnosis on the part of a resident invariably allows the surgeon ample time to arrive at the hospital and perform the surgical operation in an interval of time, which is life-saving to the patient. The beneficial results obtained by early operation are well recognized. It tends to lower mortality and postoperative complications, and thereby lessen the period of hospitalization, which as noted in the foregoing study, is a matter of no mean importance.

Likewise the importance of postoperative management is evidenced by the fact that 32.8 per cent of the cases of elective surgery suffered some form of postoperative complication, of which upper respiratory and chest involvements played a significant role; if this percentage could be lessened, the postoperative course would be much smoother, the mortality lower, and the financial liability on the part of both the patient and hospital proportionally reduced.

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There are four methods by which collapse of the lung may be obtained:

1. Collapse of the lung alone by artificial pneumothorax.

2. Collapse of the lung and of the chest wall by thoracoplasty.

3. Local collapse of the lung and of the parietal pleura—extra-pleural pneumolysis.

4. Partial collapse, mainly of the lower lobe, by evulsion of the phrenic nerve, producing thereby hemidiaphragmatic paralysis.

From—"Surgery of the Lung and Pleura" by H. Morriston Davies, M.D.

ETIOLOGY OF APPENDICITIS

F. Gregory Connell, M.D.

OSHKOSH, WISCONSIN

AMONG the many causative factors of appendicitis, "obstruction" to emptying of the appendix, seems to be rational and is becoming more generally accepted.

Appendicitis may be, and often is, a result of the following sequence: (1) obstruction with resultant, (2) increase in contents (hypersecretion, growth of bacteria and their products) which is followed by (3) distention with consequent (4) interference in circulation which allows (5) infection from the septic lumen (with nonseptic lumen a type of mucocele might result).

This infection may be confined to the mucosa, or involve the musculature and may or may not, extend to the serosa with or without perforation. When the catastrophic consequential complications, perforation and peritonitis, occur, the treatment then must be directed chiefly against the peritonitis.

A sharp distinction between appendicitis *per se* and its complication, perforation peritonitis, as exists between peptic ulcer and its perforation peritonitis, will promote better understanding of the disease.

But in treatment, as there are no reliable indicators telling which case will be and which will not be so complicated, the early diagnosis of acute appendicitis should be followed automatically by appendectomy to prevent the development of the serious and often fatal complication, perforation peritonitis.

Interference with emptying of the appendix may be due to: A. Foreign body, B. Stricture, C. Functional derangement.

A. Foreign body (fecolith, concretion), inflammatory reaction (mucous plug, edema) or neoplasm;

B. Stricture, stenosis, deformity, angulation or kinks may occur at various points in the appendix, due to excessive or imperfect fusion of the parietal and the visceral peritoneum following either inflammation or developmental defects in migration, rotation and descent of the cecum. The angle at which the appendix and the cecum unite may be instrumental in promoting or interferring with proper drainage. The modus operandi of A and of B is quite obvious, but that of C calls for elucidation.

C. Functional derangement: circulatory or nervous imbalance, hypotonicity or hypertonicity—spasm—of the neuromusculature may presumably occur at the cecoappendiceal junction, where a definite "valve" was described by Gerlach in 1847. The existence of a true "sphincter" at this location has been denied, but the necessary ingredients for such a mechanism are present; viz., circular muscle with intrinsic and extrinsic nerve supply. Barium and *x*-ray studies may demonstrate hypertonicity and hypotonicity at the adjacent ileocecal junction (Bauhin's valve) but at the appendicocecal junction such a demonstration is not equally satisfactory. Krenn, claims to have demonstrated by roentgenologic studies, that spasm of the ampullar portion of the appendix may promote retention of feces in the distal portion. Westphal (also quoted by Cutler) found that activity of the vagus caused a shortening of the organ and constriction of the proximal or ampullar portion of the lumen.

Because of the striking similarity between the pylorus (with its adjacent cul-de-sac, the biliary tract) and the ileocecal valve (with its adjacent cul-de-sac, the appendix) one would seem justified in assuming that the recognized dyskinesia (Ivy) and dyssynergia (Best), i.e. disturbed motility due to abnormal movements associated with change in muscle tone, of

the sphincter of Oddi, might possibly likewise occur at the appendicocecal junction.

Normal visceral function depends upon subconscious, coordinated, synchronized and mutually antagonistic neuromuscular reflexes dependent largely upon autonomic nervous system. If they are incoordinated, asynchronized or in disproportion, the resultant function is abnormal and is interpreted by the individual as "complaints," that serve the physician as "symptoms" in his study of the case.

Plainly evident cardiospasm or pylorospasm and congenital pylorospasm in the *proximal*, and "spastic" sigmoid and Hirschsprung's disease in the *distal* alimentary tract, suggest the possibility of similar dysfunction in the *middle*, or ileocecal, region of this tube.

Spasm is recognized as an etiologic factor in ulcer of the duodenum.

If spasm is effective in creating an ulcer (an infection atrium) in the proximal gut; there is reason to think that a similar spasm in the midgut might also create an ulcer, the different clinical course in the two locations being due to differences in:

Hydraulics: one: open at both ends. Other: closed at one end (cul-de-sac).

Contents: one: plus Hcl, comparatively aseptic. Other: septic.

This possibility is increased when one recognizes the fact that the autonomic nerve supply of this particular region of the gastrointestinal tract is strikingly different from all other regions in having a double instead of the usual single parasympathetic innervation.

The sympathetic nerve supply originates from a single locality, i.e., the lower dorsal and upper lumbar paravertebral ganglia. The parasympathetic nerve supply, on the contrary, originates from two sources, one in each extreme, the bulbar and the sacral, of the central nervous system. This difference may have an influence upon symptoms referable to the right iliac fossa before and after appendectomy.

Despite the abundance of clinical and autopsy material, the nerve supply of the region of the appendix has not been definitely determined. It has been claimed that the splanchnic vessels have no para-

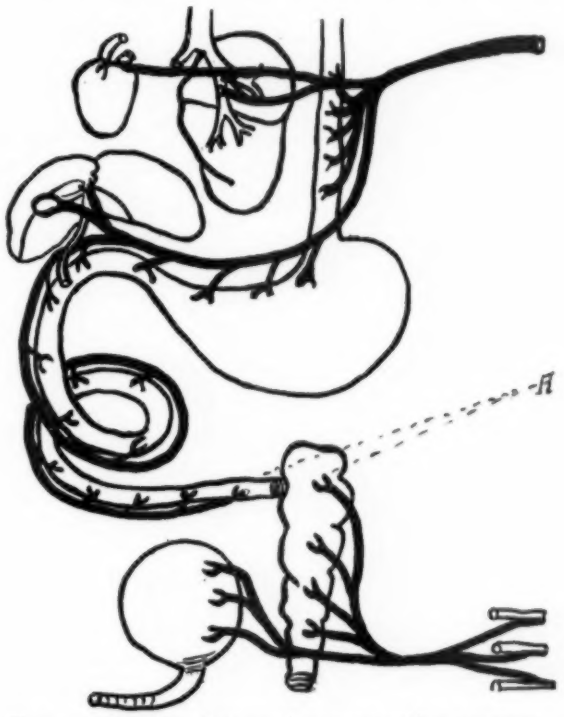

Fig. 1. A. Region of normal, over or under, parasympathetic innervation. (From W. H. Gaskell; "The Involuntary Nervous System," 1916. Longmans, Green & Co., p. 25.)

sympathetic innervation (Fig. 1) Kuntz finds, "a few such fibers" but states (p. 217), "The exact distribution of the efferent vagus fibers to the large intestine, in man, is unknown."

Livingston[1] says, "The exact portion of the colon at which the vagus innervation ceases and the sacral parasympathetic influence begins is not known. It was thought formerly that the vagus influence ended at the ileocecal valve, but Garry and Schmidt have demonstrated degenerated fibers as far as the distal part of the transverse colon, after section of the vagi."

The sacral innervation of the parasympathetic (the pelvic nerve, the "nervi Erigentes") is usually described as terminating at the midtransverse colon, but some authorities (Gaskell, Langley and

[1] Livingston, W. K. Clinical Aspects of Visceral Neurology, Charles C. Thomas, Springfield, Ill., 1935, p. 9.

Andresen) found it at times to extend to the entire colon and include the cecum and appendix.

Postmortem study of the gastrointestinal neuromusculature in cases of previous lumbar sympathectomy might help to solve the problem.

The acute obstruction must, of course, be considered separately from the chronic obstruction, i.e., "obliteration" of the lumen of the appendix which usually starts at the tip and proceeds to the base.

The most interesting claim of Masson (1931) that autonomic nerve proliferation is largely responsible for this obliteration and that neuromata formations are largely responsible for the often coincident symptoms has been confirmed by a few. But in the most recent contribution bearing upon this subject Collins,[2] says "Many types of nerve tissue were used before reaching the conclusion, in no specimen could Masson's contentions be substantiated."

Under normal conditions it may be presumed that the peripheral *lower* distribution of the *upper* parasympathetic (vagus) nerve supply and the peripheral *upper* distribution of the *lower* parasympathetic (pelvic) nerve supply meet and unite to form a single contiguous and continuous innervation that is antagonized by the single sympathetic innervation.

Recently Cannon and others have shown that stimulation may be humoral, that actual contact of nerve and cell is not essential to function. This has been shown by continuance of function after complete denervation and even after devascularization, as is seen in the automatism of the isolated heart muscle and of the intestinal neuromusculature.

The schematic drawings and the following tabulation of Livingston[3] are most helpful in showing the autonomic reflexes that may be initiated by irritation at or near the appendix.

The spinal reflexes are: skin hyperesthesia, umbilical and right abdominal (lower thoracic and upper lumbar nerves); and vomiting (phrenic and intercostal nerves).

Under abnormal conditions this divided parasympathetic nerve supply, by failure of the terminal fibers to contact or by overlapping, may result in defective or excessive innervation.

Failure of proper anatagonism to the single and more constant sympathetic, by the double, more variable, parasympathetic, with resulting disequilibrium might cause irregular function with consequent local or transferred symptoms.

Maldevelopments such as nevi, clubfoot, cleft palate, extrophy of the bladder, spina bifida, supernumerary digits or syndactylism, variable number of vertebras, etc., are plainly evident. Such abnormalities and the complicated embryological development makes plausible the occurrence of variations in the more minute anatomical structures.

Reflexes	Sensory	Voluntary Motor	Involuntary Motor	
			(Somatic)	(Splanchnic)
Parasympathetic.....	Headache Nausea	Hoarseness Neck muscle spasm Vomiting	Salivation	Bradycardia Gastric hyperactivity Intestinal spasticity Colicky pains.
Sympathetic.....	Cutaneous hyperesthesia	Muscular rigidity	Vasomotor changes	Inhibition of motion in ileocecal region.

[2] Collins, D. C. *Ann. Surg.*, 104: 199 (Aug.) 1936.

[3] Livingston, Edw. "A Clinical Study of the Abdominal Cavity and Peritoneum," 1932. New York. Hoeber

Less apparent, obscure, invisible, microscopic anatomic irregularities or variations, such as excessive or defective parietovisceral fusions with varying organ fixations, or abnormalities in the small vascular or nerve distribution in urinary tract or in muscular development (congenital pyloric stenosis or Hirschsprung's disease) are not uncommon and may be due to developmental errors.

Variations in the autonomic innervation might possibly be factors in so-called idiosyncrasies, instabilities, susceptibilities or in "constitutional inadequacies."

One type of continuity of the two parasympathetics might be found in the sthenic, and another in the asthenic, individual.

Differences between the parasympathetic and the sympathetic nerves may be divided into anatomic and physiologic.

The anatomic may be macroscopic or microscopic.

Macroscopic. A fundamental, though not absolute, difference in the two divisions of the autonomic, is the relative lengths of the preganglionic and postganglionic fibers.

The *parasympathetic* preganglionic fibers are long, from the center to the intramural ganglia in the wall of the viscera; the postganglionic fibers are consequently short.

The *sympathetic* preganglionic fibers are shorter, from center to the intermediate ganglia; the postganglionic fibers are therefore longer from the ganglia to the viscera, except those to the suprarenal gland, that end by synapsing with the intrinsic ganglionic cells of the medulla of that organ.

Differentiation is comparatively reliable at the origin and in the proximal larger fibers.

The *parasympathetic* (vagus and nervus erigentes) may be traced from the bulbar and sacral origins to their smaller terminal filaments.

The *sympathetic* may be followed from their origin in the paravertebral ganglia, through the abdominal (semilunar mesenteric, etc.) plexuses, and for a longer or shorter distance of postganglionic fibers, to their smaller terminal filaments.

At the terminal network of peripheral distribution, a clear cut differentiation is extremely difficult, if not impossible. The final distribution of the *parasympathetic* is in Auerbach's plexus between the longitudinal and circular musculature and by very short postganglion fibers to the final cellular termination.

The final distribution of the *sympathetic* is in Meisner's plexus situated between the submucosa and the mucosa.

The *microscopic* differences are based upon the presence or absence of a myelin sheath surrounding the axis-cylinder. As a rule the preganglionic fibers are myelinated and the postganglionic fibers are unmyelinated (a few postganglion fibers acquire delicate myelin sheaths).

Theoretically, by staining the myelin sheath it would seem to be possible to identify and to estimate the number of myelinated and nonmyelinated nerves. In the mesentery or the gastrointestinal wall proximal to the plexuses of Auerbach and Meissner, the medulated should be parasympathetic and the nonmedulated should be sympathetic nerve fibers. Distal to the intrinsic neuromusculature both systems would, of course, be nonmedulated. But as this medulation and nonmedulation are not always constant such a method is probably impracticable[4] (Kuntz p. 425). Geist[5] says there would be no method of distinguishing between medulated motor and sensory fibers by staining the myelin sheath.

Physiologic. An attempt has been made to differentiate the nerve cell of the ganglia into: Type I. Vagus and Motor; Type II. Sympathetic and Inhibitory. (Dogiel and Hill, Kuntz, p. 224.)

It has been found that gastrointestinal motor and secretory activity seem to be due to an intrinsic neuromuscular mechanism, myenteric (Meissner) and submucous

[4] Kuntz, Albert, A Textbook of Neuro-anatomy. Lea & Febiger.

[5] Geist. U. of Wisconsin, personal communication.

(Auerbach) plexuses, which are automatic and self-sufficient, but are controlled or regulated by the extrinsic parasympathetic and sympathetic nerves.

The intrinsic neuromuscular mechanism has been likened to a horse and buggy (a satisfactory motor unit) and the two divisions of the autonomic nervous system to the reins that exert control.

These intrinsic plexuses have been found to differ in structure in different locations, for example, the myenteric plexus in the stomach is predominantly vagus, while in the intestine it is about equally vagus and sympathetic. The submucous plexus in the stomach is about equally vagus and sympathetic, while in the intestine the sympathetic is predominant.

"More knowledge regarding the composition of the inter cellular plexuses in the enteric ganglia awaits further investigation." (Kuntz p. 227.)

The functional differences have been studied by observing the responses to pharmacologic endocrine, vitamine or other substances.

Other factors that have been found to play an important role and to lend confusion in the functional balance or imbalance of the autonomic nervous system are: Acid/Base: K/Ca; O/Co_2: Colloid/Crystalloid}proportion in body fluid.

It has been quite definitely determined that adrenalin, ephedrine, cocaine, tyramine thyroid, and hypophysin stimulate the sympathetic. Ergotamine and ergotoxine depress the sympathetic. Physostigmine, pilocarpine, muscarine and cholin derivatives, and acetylcholine stimulate the parasympathetic. Atropine, tyrosine, and homatropine depress the parasympathetic.

It has been stated as a general rule that the *vagus* usually promotes contraction (motility) and the *sympathetic* causes relaxation (inhibition) but under certain conditions such effects may be reversed. This confusion has been explained by the assumption that the vagus may include some inhibitory fibers and the sympathetic nerves may include some motor fibers. Another explanation lies in the observation that the type of response depends upon the initial tonic state of musculature; for example: In the stomach, vagus stimulation causes contraction if the organ is atonic, and relaxation if it is tonic or hypertonic (or vice versa); in the intestine the vagus stimulation causes relaxation, if the bowel is atonic, and contraction if it is tonic or hypertonic (or vice versa).

Discrepancies, inadequacies and lack of knowledge characterize the present day interpretation of the function of the autonomic nervous system.

This seems to be especially true of sphincter action, but it would seem that the vagus contracts the pylorus and dilates the ileocecal sphincter, while the sympathetic dilates the pylorus and contracts the ileocecal sphincter.

Many experimental observations have demonstrated the influence of autonomic nervous system upon gastric, motor and secretory functions.

The clinical observations of Cushing, Comroe, Grant and others have served to emphasize the neurogenic or psychogenic factor in the etiology of ulceration of the upper gastrointestinal tract.

Abnormal stimulation of the centers, tumor or trauma, of the diencephalon, or irritation of the nerve trunks or terminals may produce "symptoms." It has been proved that these functional disturbances may, if continued, be followed by organic lesions such as congestion, edema, inflammation, erosion and sometimes ulceration of the gastroduodenal mucosa that resembles peptic ulcer.

Whether such lesions are, or are not, typical peptic ulcers is not entirely settled.

There seems to be no rational reason why such changes should occur in the upper, or gastroduodenal region and why similar ones should not occur in the lower, or ileocecal region.

Differences in the characteristics of the tube itself, the appendix, a cul-de-sac, the external relationships and the character of the contents (sterile and acid in the

upper, septic and alkaline in the lower) may partly account for the striking differences in the local effects, and the frequency of complication and accidents in the lower, or ileocecal as compared with the upper or gastroduodenal, areas.

The not uncommon occurrences of so-called "epidemics" of "appendicitis" in hospital nurses, students or interns, or in families following an operation in one member, may possibly be explained upon such a psychogenic or neurogenic basis.

Autonomic imbalance in the appendicocecal neuromusculature *with* obstruction, may be one cause of acute appendicitis; *without* obstruction it may account for the cases in which symptoms have not been satisfactorily explained by the operative findings.

If this reasoning is logical it would be a natural reaction to look for a similar autonomic nervous disturbance in another region of the gastrointestinal tract.

The pylorus, the gastroduodenal junction, at the opposite (upper) end of the small bowel is notoriously subject to motor dysfunction, which with the *x*-ray barium meal is subject to actual visualization and study.

In this location one finds conditions relative to parasympathetic and sympathetic innervation, strikingly different and yet similar with potentialities for imbalance.

Here the parasympathetic is seemingly predominant with the pyloric branches coming from the middle, and not the extremity of the nerve, in contrast to the situation at the ileocecal region.

The sympathetic, on superficial gross examination seems equally distinct and definite, but on tracing the preganglion fibers back to their origin in the paravertebral ganglia, one finds a condition in which slight anatomical variation might result in over or under sympathetic innervation.

Under normal development the semilunar and mesenteric ganglia are supplied by the splanchnic nerves from the lower, the sixth to twelfth paravertebral ganglia.

The other (upper) six ganglia supply the stellate ganglia and the cardiac nerves.

This arbitrary division into upper (cardiac) and lower (mesenteric) thoracic nerves is the usual, or average finding, but may not be constant.

With abnormal development it is conceivable that the cardiac nerves might receive more or less branches and that the splanchnic nerves might, in consequence, receive less or more branches, from the nerve trunk.

And when it is remembered that even the number of vertebras may vary in different individuals, variation in origin and course of spinal nerves does not seem impossible.

Such abnormalities in the origin of the cardiac and the splanchnic nerves might explain, not only disturbances in gastric or cardiac function but also the close relationship of thoracic and abdominal symptoms. Under such circumstances one finds at the pylorus potentialities for autonomic nervous imbalance analogous to those at the ileocecum.

But with this striking difference, at the pylorus, the parasympathetic, when compared with the sympathetic innervation, appears to be the more constant factor; while at the ileocecum it is the more inconstant (either plus or minus).

Difficulty in differentiating between symptoms of pyloric spasm and adjacent biliary tract dysfunction is well recognized and is probably partly due to their similar nerve supply.

Similar difficulty might be experienced in properly interpreting symptoms due to ileocecal and adjacent appendicocecal dysfunction, again due, in all probability, to a similar nerve supply. Proof of function or dysfunction of the ileocecal valve is more or less demonstrable by roentgenograms and other means, but similar proof concerning the appendicocecal communication is extremely difficult, though in a recent contribution Galambos[6] discusses the sub-

[6] Galambos, A. *New York Am. Jour. Digestive Dis. and Nutrition*, 3: 413 (Aug.) 1936.

ject of "Functional changes in the vermiform appendix producing diverticular saculation of the spastically contracted organ" with a series of *x*-ray photographs.

It would seem entirely justifiable to assume that disturbance in autonomic nervous balance might cause spasm or hypertonicity of the neuromusculature at the appendicocecal juncture, which might help to answer the original question as to the why of the obstruction that caused a certain proportion of cases of appendicitis.

Possibilities of prophylactic therapy of course, present themselves, but in closing let me repeat (paragraph 3 p. 1).

SUMMARY

Attention is directed to the existing confusion concerning the cause of appendicitis.

Distinction between appendicitis per se, and perforation and peritonitis, is important.

Appendicitis is considered as a result of the sequence of obstruction, distention, circulatory stasis and infection.

"Obstruction" is recognized as a reason but this prompts an inquiry as to the cause of the obstruction.

Obstruction may be due to:

A. Foreign body: mucus plug, edema or neoplasm;

B. Stricture: stasis, angulation, deformity;

C. Functional derangement: spasm.

The modus operandi of A and B is quite obvious, but that of C calls for elucidation.

The striking difference in the ileocecal region from all other gastrointestinal situations is that the parasympathetic distribution is double, which permits the possibility of overinnervation or underinnervation.

Sympathetic-parasympathetic imbalance is a possible cause of hypertonicity or hypotonicity at the cecoappendix juncture. The existence of a true sphincter (Gerlach's valve) at this point has been denied, but circular muscle and extrinsic and intrinsic nerve supply, the necessary ingredients, are present.

Malfunction in the foregut and hindgut, cardiospasm and pylorospasm, and colon and rectal spasm, are distinct clinical entities and similar disturbance might rationally take place in the midgut.

Difference in hydraulics, one a cul-de-sac the other with a double open end, may account for some of the clinical differences.

Dyskinesia of the sphincter of Oddi, adjacent to the pylorus, is recognized; similar dysfunction at the appendicocecal junction, adjacent to ileocecal valve, is theoretically plausible.

Difference in contents, one septic the other comparatively sterile, may partially account for different clinical courses.

It would seem entirely justifiable to assume that disturbance in autonomic nervous balance might cause spasm or hypertonicity of the neuromusculature at the appendicocecal juncture, which might help to answer the original question as to the why of the obstruction that caused a certain proportion of cases of appendicitis.

To be consistent we must offer an explanation for this autonomic imbalance; and with our present knowledge, it would seem very probable that overparasympathetic or underparasympathetic innervation of the ileocecal region is due to variations in embryologic development.

PROCIDENTIA: A NEW OPERATION FOR CURE OF FOURTH DEGREE PROLAPSE

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IN 1919 when I published an article on the Watkins-Wertheim interposition operation the technique was quite new and was performed by only a few surgeons. It passed through the usual cycle of many new procedures, i.e., the attempt to give it too broad an application, and for that reason a very valuable and worthwhile treatment for the "procidentia syndrome" was discontinued by some and even condemned by others.

Fortunately, those of us who were able to gain more experience with the operation, to analyze the mechanism of the procedure and study the indications and limitations, were well repaid for our efforts, for at the present time the consensus of opinion among gynecologists is that there is no other surgical procedure which obtains the results of the Watkins-Wertheim interposition operation within its limitations. The limitations and indications are treated later in this paper.

In discussing this new operation for procidentia "vaginal subtotal," I shall not excerpt from the voluminous material except to say that the literature has been searched quite thoroughly back to 1850 and nothing has been found describing it. On this basis I believe it to be entirely new and original, and in our clinics it is now known as the "Chaffin vaginal subtotal" for prolapsed uterus. I shall first take up the mechanism[1] of the uterus, bladder and cul-de-sac descensus.

The etiology, with rare exceptions, is the trauma of childbirth. The right angle axis of the uterus to vagina is changed to an obtuse and finally parallel axes. Associated with this change of the relationship of the axes, is a stretching of the round and broad ligaments, and lack of support. The intra-abdominal pressure is at once placed on the only remaining structural support, the sacrocervical ligaments, and when these are stretched excessively, prolapsus occurs. (I have substituted the term sacrocervical to the anatomical sacrouterine.) It is needless to say that the predominating symptom in this affliction of woman is that of "residual urine bladder symptom." The anterior space is increased, anterior vaginal fascia torn and the bladder descends with or without the uterus.

It is not uncommon to find the bladder "slid down" 1 to 2 inches on the anterior wall of the uterus. This produces a residual and drags on the bladder neck, a firmly fixed point. If the descensus of the uterus is only the first or second degree, not yet appearing at the vulva while sitting or squatting, and the bladder bulging to any degree, the Watkins-Wertheim interposition operation is the ideal operation to hold the bladder up and is superior to all other methods. My experience and the experiences of others has convinced me that the anterior wall fascia cannot be "identified" and plicated to make a sufficiently strong support to hold a bladder filled with urine (1 pint to 1 pound in weight). The old elliptical excision of the anterior wall and longitudinal suture is practically useless for the vaginal wall has no tensile strength and will immediately stretch to its former width. Also, in this unscientific operation the suture of the vaginal wall produces a corresponding infolding of the bladder wall, not in itself a good thing to do. The inter-

[1] KOSTER, H. Support of the pelvic viscera and the mechanism of prolapse. *Am. Jour. Surg.*, 29: 226–235, 1935.

posing of the uterus completely closes the opening, leaving only the urethra emerging and the bladder can never again appear.

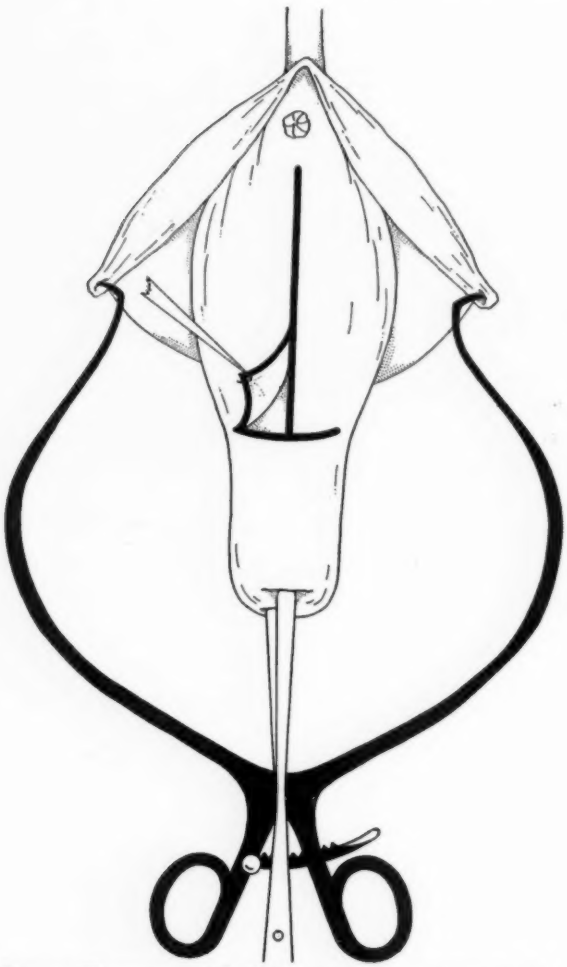

Fig. 1. Exposure with Gilpi retractor, uterus pulled down with tenaculum sound and dissection begun.

Incidentally, this operation will remedy most of the "dribbling" or slightly incontinent cases by taking all strain and drag from the bladder neck. The reason for this relief from dribbling is easily understood when we recall that all "sphincter" muscles must be a perfect circle and be centripetally active to be effective. "Drag" on the bladder neck in cystocele, changes the sphincter from a circle to an ellipse and therefore leaking occurs.

The technique of this operation is too well described in many textbooks and journals to dwell further on it except to emphasize again that it should be done not only in the first and second degrees of prolapsus uteri but in any and all cystoceles and bladder hernias. The only exceptions I make to this rule are in those cases in which previous pelvic (abdominal) surgery, as ligament shortening, has been done or in cases of pelvic inflammatory disease or adhesions preventing the delivery of the uterus. In these, I do what I think is the next best operation and that is the advancement or replacement operation.[2] This replaces the bladder at a higher level on the anterior uterine wall and if there is no prolapse the results are satisfactory. In passing, I will say that future pregnancies with the Watkins-Wertheim interposition operation, is contraindicated and that is disposed of by salpingotomy or a postmenopausal operation.

We will now take up the subject of the complete procidentia beginning where we left off, with the limitations of the Watkins-Wertheim interposition operation, and include everything down to the *n*th degree.

To the analytical and mechanical minded surgeon, it is at once seen that the condition cannot be scientifically corrected by any "hanging up" operation by the abdominal route which includes such procedures as Murphy, Kelly and others which are present, practically discontinued.

Reasons. 1. The abdominal wall is the only fixed point to which to "tie" and this type of operation is unanatomical, unphysiological and most of all so mechanically incorrect that the patient is not only unrelieved but often times made worse by adding discomfort greater than the original.

2. The bladder is a cystic organ and must be supported from below and *not* "*bung.*" I shall mention total vaginal hysterectomy only to condemn it, as I have since my student days, considered it a destructive rather than a reconstructive operation and not in the least an operation for the cure of procidentia.[3,4] Its indications

[2] Jones, Walter C. Cystocele, replacement of the bladder. *Southern Surg.*, 181, 1934.

[3] Palmer, A. C. The prolapse syndrome. *Brit. Med. Jour.*, 11: 899, (November) 1934. "Vaginal hysterectomy formed no part of the operation of prolapse . . . in removal of cervix, loose the last stable suture point."

should be restricted to the removal of the pathological uterus.

In the few I did in my early experience, I always felt guilty and incompetent in removing displaced or protruding organs and tissue inasmuch as I did not know what else to do with them. This feeling is now even stronger when I sit in an operating room and witness the best of surgeons or gynecologists do a total vaginal hysterectomy. Because of this guilty feeling, I set about to analyze the mechanics of procidentia and a constructive and reconstructive surgical treatment. I will endeavor to lay this analysis before my reader with word description and diagrammatic drawings.

We will start with the matter of the sacrocervical ligament. In procidentia, these structures (although the last to give away) are immensely elongated or their function is destroyed altogether. Therefore, the Watkins-Wertheim interposition operation will not cure this patient as this operation deals only with anchoring the fundus forward beneath the urethra but does not hold the cervix back towards the sacrum. This lesson we learned in the early cases and the patient returned with the bladder well supported and all bladder symptoms relieved *but* the cervix appeared at the vulva and "slipped" over the reconstructed perineum. Now what to do to keep the cervix back? Sometimes we could reef (by the abdominal route) the sacrocervical ligament to assist us. In others we attempted to bring the broad ligament anterior to the cervix at the lower edge but this was difficult and unsatisfactory and was gradually abandoned.

Some gynecologists (Phaneuf)[5] go into the belly especially to reef the sacrocervical ligament after completing the usual Watkins-Wertheim interposition operation. The reason this latter treatment did not result in adequate support is that those much

Fig. 2. Anterior vaginal wall dissection completed high and well lateral, and bladder mobilization begun.

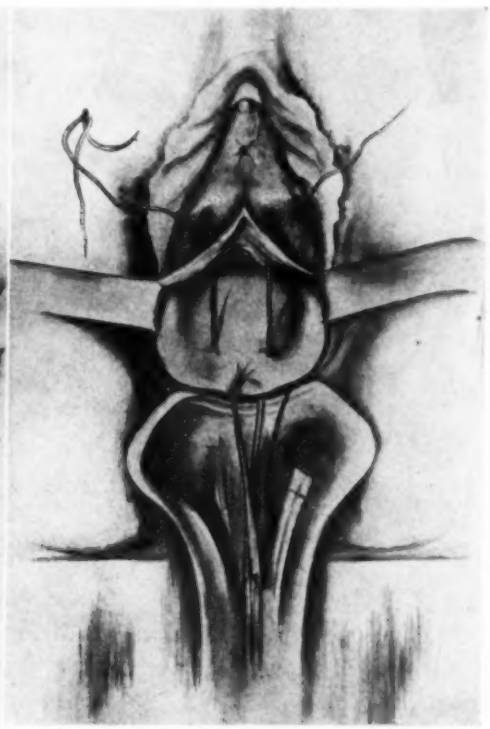

Fig. 3. Fundus being delivered beneath bladder, preparatory to its removal.

He further states he was unable to see through because there was no way to "hitch the cervix up."

[4] Shaw, N. N. Cystocele. *Jour. Surg., Gynec.,* and *Obst.,* 55, 1934. Shaw mentions the matter of prolapse by stating, "Vaginal total hysterectomy, usually done but some operators retain the cervix which gives a better anatomical and functional result."

[5] Phaneuf, L. E. Surgical management of prolapse of uterus and vagina. *Surg., Gynec. & Obst.,* 63: 385–390, (September) 1936.

over-rated sacrocervical ligaments are little, if anything more, (in the overstretched) than thin folds of peritoneum, of the tensile

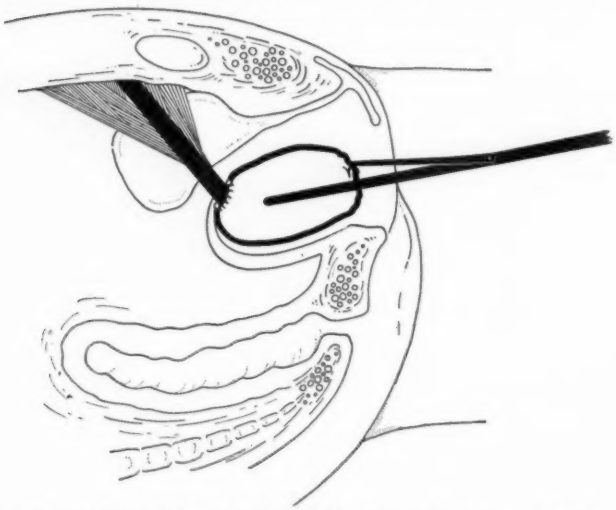

Fig. 4. Remaining cervix held in tenaculum sound, and round and broad ligaments have been sutured into posterior surface at the posterior vaginal attachment.

strength of adhesions. Now, in reviewing the technique of the vaginal hysterectomy, particularly the one known as the "Mayo" some modification of which is usually done at present, we find that there is an important support available in the absence of the sacrocervical ligament and that is the round and broad ligaments.

These are so well recognized that in the operation of total vaginal hysterectomy they are imposed upon to support the bladder after the principles of the Watkins-Wertheim interposition operation, by suturing together median below the urethra and "interposing" (identical technique of the Watkins-Wertheim interposition operation of the corpus uteri). The Mayo and other vaginal hysterectomy operations not further abuses, or imposes, upon the round and broad ligaments by attaching, as well as possible, the mutilated shortened vaginal canal to the broad ligament. This is a great deal to expect of these frail structures (recall the anatomical structure of the broad ligaments). As I look at the whole matter, I was impressed with the fact that the cervix and uterus were not at fault in the prolapsus, so why not leave them intact as far as possible and attempt to support them? The vagina with the cervix remaining is a much more substantial structure to support or "tie up," or "plug up" the pelvic outlet than a ragged muti-

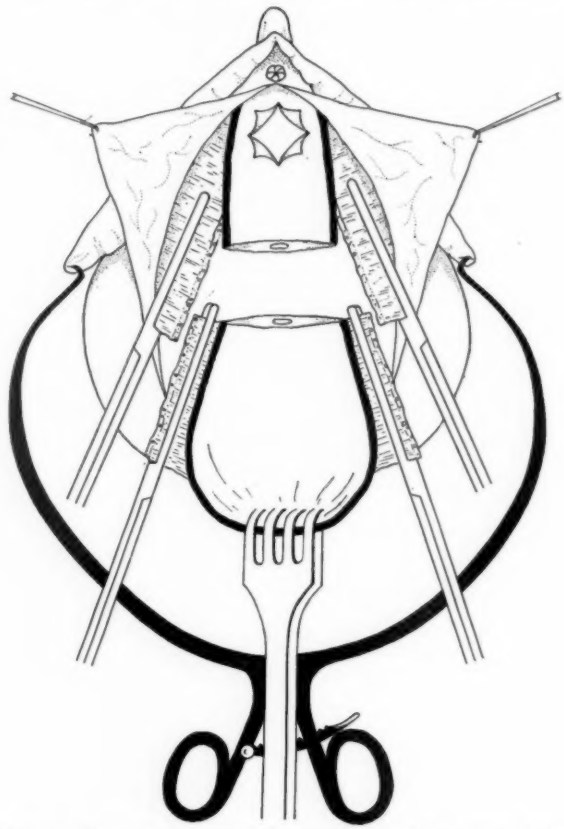

Fig. 5. Corpus amputated, cervix has been denuded on posterior surface to attach the broad and round ligaments.

lated vagina with the cervix removed, and functionally gives the patient a better anatomical and physiological genital canal. Remembering that the anchor suture of the interposition operation always holds the fundus up and the bladder back, I set about to find some way to keep the cervix pulled back towards the sacrum. I remembered the round ligament. I was at first impressed that the pull from the internal ring was not in the same line as the sacrocervical ligament but hoped it would be in near enough line to answer my problem. Now how to make the round ligament available? This is done by the "Chaffin" vaginal subtotal hysterectomy. The *corpus only* is removed through the anterior space, thereby making the round ligament available. The round ligament, with the broad ligament tied en masse, is transferred to the

lower posterior cervix or even into the vaginal wall, cul-de-sac tissue, and then sewed firmly into a denuded area. The remaining cervix after high amputation of the corpus, is usually as long as the average entire uterus in the post-menopausal patient with first to second degree prolapse, is treated exactly as the entire uterus is in the Watkins-Wertheim Interposition Operation.

Step by step, the technique in detail follows:

Begin as in the interposition operation, using a Gilpi retractor tenaculum sound (Fig. 1).

1. Incise over bladder, desecting vaginal wall well back (Fig. 2).
2. Mobilize bladder, pushing up and incise peritoneum (Fig. 2).
3. Remove tenaculum sound.
4. Grasp fundus with tenaculum and deliver (Fig. 3).
5. Clamp broad ligament and high amputation of corpus, exactly the same as the technique for abdominal hysterectomy (Fig. 5).
6. Ligate broad ligament and other bleeding points.
7. Insert tenaculum sound in "cut end of cervix" (internal os) and "throw vaginal wall" (cul-de-sac tissues) up into view by traction and depressing handle of tenaculum sound (Fig. 4).
8. By transverse or crucial incision (to scarify) in cul-de-sac tissue (posterior vaginal wall) a point of implantation is made for the round ligaments (Figs. 4 and 5).
9. Suture the ligament well into the prepared area.
10. Remove the tenaculum sound and close upper end of cervix.
11. Pass anchor suspension suture through the "pubovaginal fascia" and closed end of cervix, exactly as the technique in the Watkins-Wertheim interposition operation of interposing the intact uterus (Fig. 3).
12. Close the anterior incision.
13. High perineorrhaphy (Fig. 6).

CONCLUSIONS

1. A new operation "Chaffin vaginal subtotal hysterectomy" for procidentia is herein described.

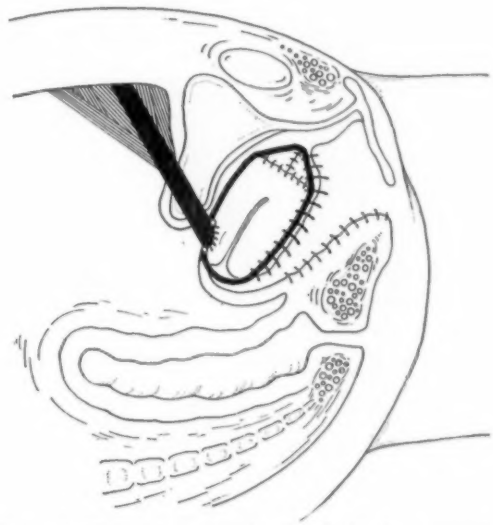

Fig. 6. Posterior cervix held back towards sacrum. Cut end of cervix anchored beneath urethra. Vaginal wall closed and perineorrhaphy completed.

2. The "keystone" of the operation is that of using the round and broad ligaments to replace the function of the sacrocervical ligament.
3. The indications and limitations of the Watkins-Wertheim interposition operation, based on the results in 200 to 300 cases, have been discussed.
4. Total vaginal hysterectomy for prolapsed uterus is herein condemned (with rare exception) and a superior substitute offered.
5. "Suspending" is not believed to be indicated in any case of procedentia.
6. This operation can be applied in all cases beyond the limitations of the interposition operation.
7. Operation is much easier to perform than a total vaginal hysterectomy and can be done in one-quarter to one-half the time.
8. The operation is far superior to total vaginal hysterectomy as a corrective measure, and leaves a normal vaginal canal.
9. Experience covers a six year period of more than fifty cases with 100 per cent physiologic and anatomic cures.

PRESACRAL NEURECTOMY IN TREATMENT OF CERTAIN PELVIC, BLADDER AND BOWEL CONDITIONS

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DES MOINES, IOWA

RECENT developments in surgery have proved that certain disturbances of the visceral system are the result of derangements of the autonomic nervous system. The present report is made upon the operative procedure of presacral neurectomy in alleviating visceral pain or adjusting physiologic dysfunction of the pelvis, bladder or bowel.

HISTORY

Jaboulay made the first attempt to relieve pelvic pain by dividing the pelvic sympathetic fibers through a retrorectal approach. A year later, Ruggi added to this investigation by advocating resection of the utero-ovarian plexus via a transperitoneal route. The results of these operations met with partial success and little further was achieved until Leriche introduced periarterial sympathectomy of the internal iliac artery as a means of relieving pain in cases of functional dysmenorrhea. The latter procedure was utilized by Cotte and Michon. In 1925, Cotte introduced the operation of resection of the superior hypogastric plexus, called the presacral nerve of Latarjet and Bonnet, for functional dysmenorrhea and reported favorable results. Cotte has listed six indications for the use of his method: (1) pelvic neuralgia, (2) vaginismus, (3) dysmenorrhea, resistant to ordinary methods adopted for its relief, (4) hypoplasia of the uterus, accompanied by insufficient and painful menstruation, (5) metrorrhagia of ovarian origin, and (6) sexual neuroses, such as masturbation, nymphomania, etc. Herman is stated to have been the first American surgeon to have performed this operation. Then a widespread application of this method was reported by many European surgeons. The same surgical procedure of resection of the presacral nerve has been employed for cord bladder, painful vesical conditions and hydroureter by Learmonth and Braasch, Ward, Douglass, Braasch and Craig, McConnell, Quinby, Bailey and Fulcher. In 1933, Abbott and Pfaff reported a case of bladder and bowel dysfunction with dysmenorrhea which responded to this operation. The relief of Hirschsprung's diseaes or megacolon was achieved by either resection of the lumbar sympathetic ganglia, or the inferior mesenteric and presacral nerves, as reported by Wade and Royle, Judd and Adson, Robertson, Rankin and Learmonth. In addition, cases of obstipation have been treated satisfactorily with this operation as reported by Rankin, Royle, Learmonth, Adson and Abbott. Since then the following American surgeons: Adson and Masson, Greenhill and Schmitz, Counseller and Craig, Abbott, White, Wetherell, DeCourcey, Bailey and Cannaday and Keene have achieved favorable results in cases of pelvic pain.

ANATOMY AND PHYSIOLOGY

The inferior mesenteric plexus gives rise to the right and left mesenteric nerves which unite below the origin of the inferior mesenteric artery to form the presacral nerve (superior hypogastric plexus). The branches from the left side pass obliquely downward, while the branches from the right side pass posterior to the artery. There are many fine anastomoses between these nerves so that a true plexus is formed which is situated anterior to the bifurcation of the aorta and is separated from it by a thin layer of fascia. The plexus then follows the course of the sacrum into the pelvis. The parietal peritoneum and a thin layer of loose connective tissue lies anterior to the plexus. The root of the mesentery of

the sigmoid colon is situated to the left. There are many variations of the superior hypogastric plexus but none of vast importance. Latarjet and Bonnet gave it the name of "presacral nerve." In about 20 per cent of the cases, the fibers join to form a new nerve, and in about 80 per cent the nerves are more or less spread out in plexus formation (Delmas and Laux). However, Elaut states that in about 2 per cent of the cases there is an arch shaped plexus.

A few filaments from the superior hypogastric plexus course along the common iliac artery. The plexus itself continues down over the promontory of the sacrum and, at the level of the first sacral vertebra, divides into two distinct nerves, the inferior hypogastric nerves. These nerves course downward into the lateral rectal space and terminate in a mass of nerve fibers and ganglion cells named the hypogastric ganglion, which is situated on the superior pelvic space between the peritoneum and the fascia covering the levator ani muscles. This plexus forms again on the anterior surface of the rectum, just posterior to the uterus and vagina, and becomes intimately connected with the uterosacral ligaments.

The nerve supply of the ovary is derived chiefly from the ovarian plexus which arises from the aortic and renal plexuses and follows the course of the ovarian artery. Hovelacque states that three fibers arise from the renal plexus to join the ovarian plexus. Other nerve bundles may be traced directly to the ovarian ganglion which is situated near the origin of the ovarian artery. There are also rami which connect with the celiac and superior mesenteric ganglia. The ovarian plexus enters the suspensory ligament of the ovary and invests both the ovarian artery and vein. These fibers divide into branches supplying the Fallopian tube and broad ligament, the hilus of the ovary and the lateral surface of the uterus. Kuntz states that the afferent fibers supplying the ovary are components of the tenth thoracic nerve.

The Fallopian tube receives fibers from the uterine plexus in addition to branches of the ovarian plexus. The afferent fibers supplying the fallopian tube, according to Kuntz, reach the spinal cord through the eleventh and twelfth thoracic and first lumbar nerves.

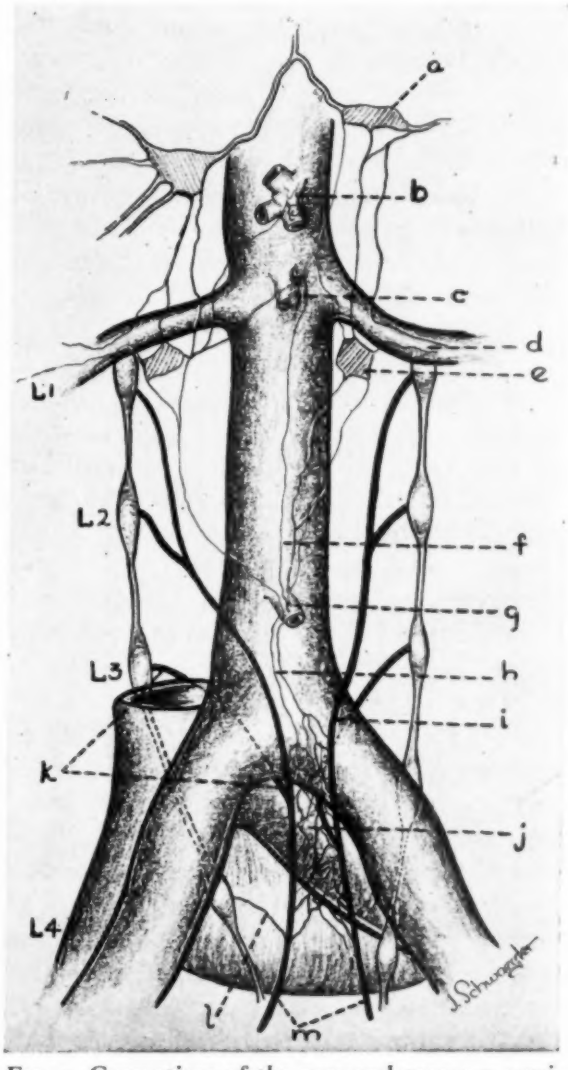

Fig. 1. Connections of the presacral nerve: *a*, semilunar ganglion; *b*, celiac plexus; *c*, superior mesenteric artery; *d*, renal plexus; *e*, renal ganglion; *f*, intermesenteric plexus; *g*, inferior mesenteric artery; *h*, middle root of presacral nerve; *i*, left lateral root of presacral nerve; *j*, presacral nerve; *k*, branch from third lumbar ganglion; *l*, branch from fourth lumbar ganglion; and *m*, hypogastric nerves (*after Laux*).

The nerve supply of the uterus includes both sympathetic and parasympathetic components. Fontaine and Herrmann state that the nerve fibers in the wall of the uterus are derived from the great plexus

of Frankenhäuser, which is situated on each side of the body of the uterus and broad ligament. This plexus receives fibers from the hypogastric plexus, the lower lumbar and sacral sympathetic trunk and the second, third and fourth sacral nerves. There is no evidence to support the view that nerve fibers terminate in the uterine mucosa, as held by older investigators.

Cleland has demonstrated, by the use of paravertebral anesthesia, that the afferent fibers supplying the uterus enter the spinal cord solely through the roots of the eleventh and twelfth thoracic nerves.

In spite of the abundant nerve supply to the ovary, there is no conclusive evidence of a direct functional innervation either of the ovarian follicle or the interstitial tissue. Undoubtedly there are vasomotor changes in the ovary as a result of this complex nerve supply, and yet Hinsey and Markee have reported ovulation induced by the injection of pregnancy urine in rabbits in the complete absence of functional nerve fibers to the ovary.

Kuntz states that the sympathetic nerves supplying the uterus and Fallopian tubes exert a motor influence and the parasympathetic nerves an inhibitory influence. However, certain experimental work has caused conflicting opinions to be held as there is evidence of uterine contractions occurring in the absence of nerve stimulation. Stimulation of the hypogastric nerves exerts a motor effect on the uterus and Fallopian tubes, but the effect may be very slight. It is also conceded that afferent fibers from the uterus and the Fallopian tubes are carried through the hypogastric plexus. Thus the removal of the presacral nerve will relieve pain, but Adson and Masson feel that interruption of efferent fibers in the presacral group also produces a dilatation of the blood vessels in the genitalia and musculature of the uterus. This operation has had no deleterious effect upon subsequent pregnancy or the normal menstrual cycle.

The bladder, sphincter apparatus and posterior urethra are supplied by three sets of nerves. The sympathetic fibers are derived from the thoracicolumbar outflow and the parasympathetic pathways arise from the sacral outflow. The pudic nerves are from the somatic system, arising from the sacral segments of the spinal cord.

The sympathetic fibers to the bladder have their origin in the first, second, third and fourth lumbar sympathetic ganglia and the intermesenteric plexus. These fibers all concentrate in the presacral nerve. More than half of the fibers in the presacral nerve are medullated preganglionic fibers which are continuations of the mesially directed branches of the lumbar sympathetic ganglia. The non-medullated fibers are continuations of the upper abdominal visceral sympathetic ganglia. Below the promontory of the sacrum, the presacral nerve divides into the hypogastric nerves as described in the anatomy of the pelvic organs. Learmonth and his colleagues observed during operations on human beings that stimulation of the presacral nerve with a faradic current produced the following results: (1) closure of the ureterovesical orifices; (2) contracture of the trigone; (3) contraction of the internal sphincter; (4) contraction of the musculature of the prostate gland; (5) contraction of the musculature of the ejaculatory ducts and seminal vesicles; (6) contraction of the blood vessels of the trigone, and (7) sensations of pain, felt in the region of the bladder. In addition, Learmonth and Markowitz studied the effect of the intravenous injection of epinephrine on the tension in a normally innervated bladder and found that the sympathetic nerves exert an inhibitory action on the expulsive mechanism of the bladder. Division of the presacral nerve will diminish the tonus of the internal sphincter of the bladder and increase the expulsive power. In the male, there is a paralysis of the mechanism of ejaculation of seminal and prostatic fluid, but no disturbance of libido or potentia. There is no effect on the sexual organs of the female and subsequent pregnancy is uncomplicated.

The parasympathetic fibers arise from the anterior primary divisions of the second, third and fourth sacral nerves. The parasympathetic fibers initiate and increase the expulsive mechanism of the bladder, inhibit the internal sphincter, convey afferent impulses for the micturition reflex and furnish part of the sensory innervation of the posterior urethra. Thus the sympathetic fibers are called the filling nerves of the bladder and the parasympathetic fibers may be known as the emptying nerves of the bladder.

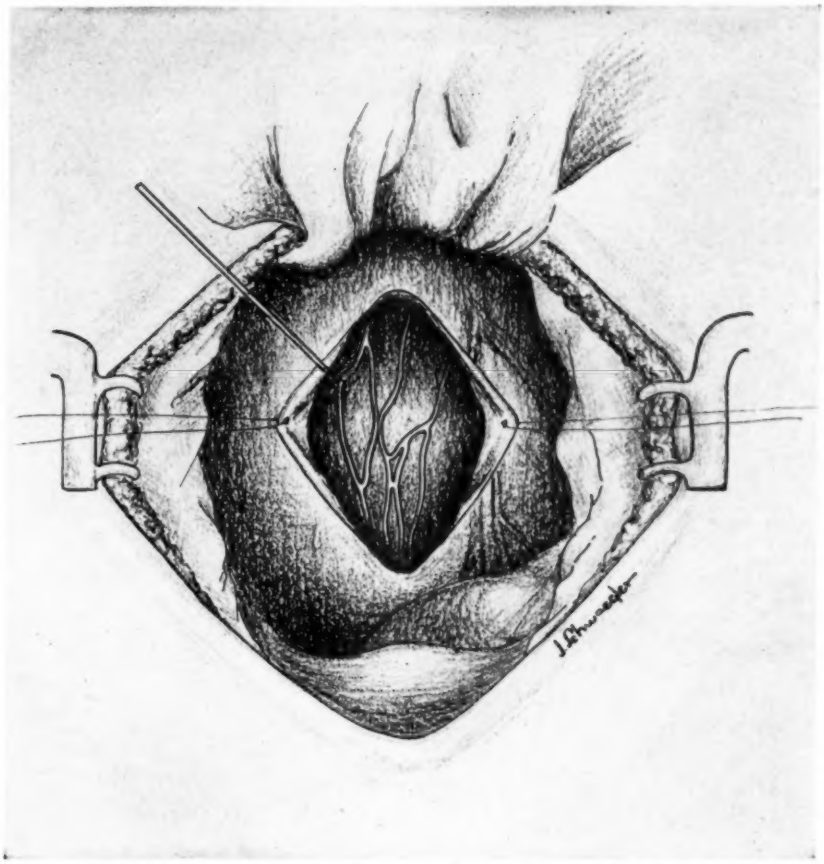

Fig. 2. Peritoneum reflected, and presacral nerve fibers picked up with hook.

The pudic fibers, which are somatic, provide motor fibers for the external sphincter and carry some sensory fibers from the posterior urethra.

The nerve supply to the lower bowel and rectum is somewhat similar to the innervation of the bladder. Gaskell has pointed out three sphincter muscles which terminate in the different regions of the primitive intestine: (1) The ileocolic sphincter at the end of the small intestine; (2) the internal anal sphincter at the end of the coprodeum, and (3) the internal vesical sphincter and the urethral muscles at the end of the urodeum. The motor nerve cells for the ileocolic sphincter are situated in the superior mesenteric ganglion, while the motor fibers to the internal anal sphincter are in the inferior mesenteric ganglion. The motor nerves to the musculature of the large intestine are part of the sacral outflow and travel through the pelvic nerve. Adson states that inhibition of the musculature of the large intestine is mediated through inhibitory nerves, the cells of which are in the inferior mesenteric ganglion; stimulation of the lumbar splanchnic nerves, or the presacral nerve, causes relaxation of the colon.

Learmonth and Markowitz have shown that electric stimulation of the presacral

nerve, and of the inferior mesenteric nerve of the colon, produces contraction of the internal sphincter of the anus. They also

the hollow of the sacrum between the two common iliac arteries. Precautions must be taken not to injure the left common iliac

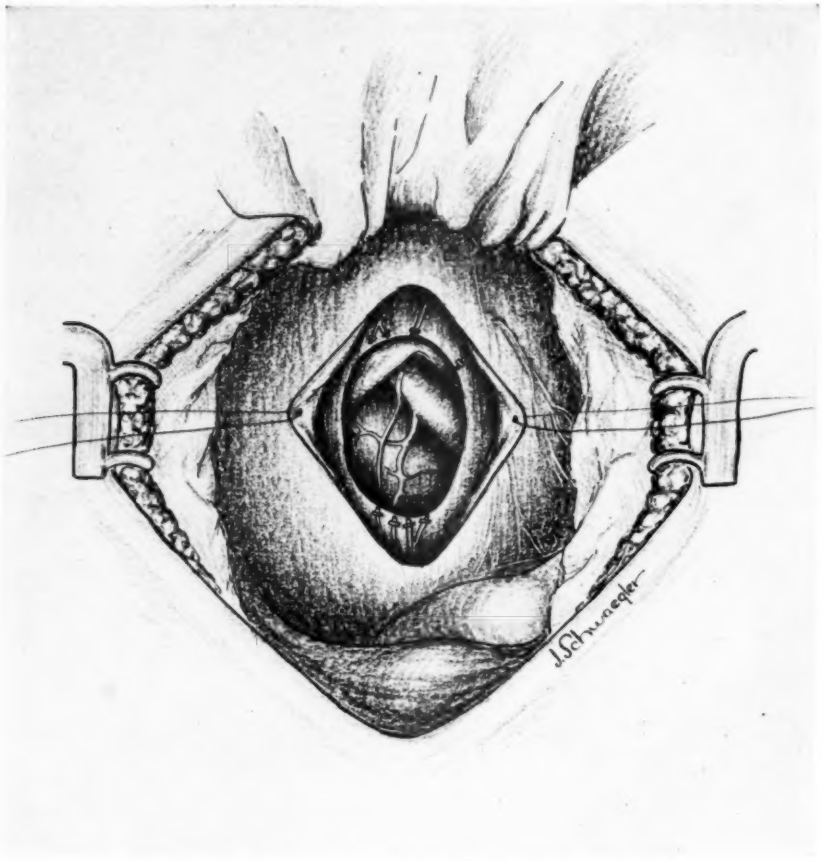

FIG. 3. Nerve fibers resected with silver clips applied.

demonstrated that division of these nerves increased the activity of the lower portion of the colon.

OPERATIVE TECHNIQUE

A low midline incision is made in the abdomen and the patient is placed in the Trendelenburg position. The intestines are packed away and a vertical incision is made in the posterior peritoneum from a point 3 cm. above the bifurcation of the aorta to 6 cm. below. The peritoneum is retracted with silk ligatures so that the superior hemorrhoidal artery is drawn out of the way on the left side. In a thin patient the nerves can be seen in a delicate plexus of pelvic lymphatics and loose connective tissue. At this point, the use of cotton pledgets will facilitate careful dissection in vein, which lies on the medial side of the artery. Just beneath the aortic bifurcation the strands of the presacral nerve can be identified and picked up on a nerve hook. The application of silver clips will prevent troublesome oozing from the vasa nervorum. The dissection is then carried down over a distance of 6 cm. so that the hollow of the sacrum is denuded. Again the application of silver clips at the end of the pedicle will prevent the seepage of lymph. The posterior peritoneum is sutured with fine catgut and the abdominal wall is closed in the usual manner.

PELVIC PAIN

Counseller and Craig have described two types of dysmenorrhea, primary and secondary. The primary dysmenorrhea

is not dependent upon pathologic change in the pelvis and many theories have been advanced regarding the cause of this painful phenomenon. The theory of retention of menstrual blood as a result of uterine displacement cannot be accepted readily because the cervix can be dilated with ease, and Novak has reported that the rate of discharge of menstrual blood of the normal woman averages about two-thirds of a drop per minute. This small amount of blood could hardly be retained by uterine displacement. The hypothesis of hypoplasia of the uterus causing pain as a result of faulty muscular development failing to respond to menstrual stimulation is not feasible because the majority of women have a uterus which is normal in size. There have not been sufficiently controlled reports to consider the endocrine system as a definite factor in the production of dysmenorrhea. It is also known that marriage and subsequent pregnancy will abolish certain types of dysmenorrhea; in addition, the psychic element must be borne in mind. However, there are certain cases in which menstrual pain is so severe that it remains adamant to any therapeutic agent and in this type of case resection of the presacral nerve is indicated.

The secondary dysmenorrhea is the result of demonstrable pathologic change in the pelvis, such as uterine myoma, endometriosis, salpingitis, ovarian tumors and marked uterine displacement. It is obvious that correction of these known pathologic processes is indicated in preference to section of the presacral nerve. At times, the recognized pathologic process seems insufficient to cause such severe pain and, in these patients, presacral neurectomy may be utilized as an adjunct in the correction of severe dysmenorrhea.

Fontaine and Herrmann have described three types of cases in which resection of the presacral nerve afforded relief from pain in 22 of 28 cases.

1. Where slight pathologic processes in the pelvis do not respond to ordinary gynecologic treatments. These periodic paroxysms of pain were described by Cotte as pelvic plexalgia. The distress begins in the region of the uterus and radiates to the anus, coccyx and urinary bladder; pain may also radiate to the back and thighs.

2. Where pain persists after correction of retroversion of the uterus or the removal of a sclerocystic ovary.

3. Where the pathologic lesion is known but which has been found to be too extensive for surgical removal; inoperable neoplasms causing severe pain.

This report deals with types 1 and 2, or those cases relating to menstrual pain associated with little or no demonstrable pathologic process.

Case 1. An unmarried woman, twenty years of age, had suffered from severe dysmenorrhea until pain had become almost constant. Roentgen therapy and currettage had failed to afford relief. The examination was negative except for a suggestive small mass in the right lower abdominal quadrant and tenderness over the sacroiliac region. Roentgenograms revealed a developmental defect of the coccyx but this was not considered to be an important factor. There was also a leucorrhea. On June 4, 1933, a currettement was performed and a small amount of reddish deciduous material was removed. Then a laparotomy was performed and the right ovary, which measured 5 by 2 by 1.5 cm. and contained a few cysts, was removed. It was felt that there was hardly enough pathologic change to cause such intense pain, so I resected the presacral nerve. The patient made an uneventful recovery and has been free from any menstrual or abdominal distress since the operation. The sacroiliac distress was alleviated to a marked extent with a corrective belt.

Although there were some organic changes, it was felt that resection of the nerve in this case was of distinct benefit.

Case 11. An unmarried woman, twenty-seven years of age, had suffered from severe dysmenorrhea since puberty, but in the past year the pain had become so severe that she was incapacitated for seven to ten days each month. In addition, she had suffered from a constant dull ache in both lower abdominal quadrants for almost a year. Examination was essentially negative except for tenderness in both lower abdominal quadrants. On May 15, 1934, under

avertin anesthesia, laparotomy was performed and the pelvic organs were found to be normal in size and appearance. The presacral nerve was resected and the patient made an uneventful recovery. When seen August 2, 1934, the patient stated that the menses had been painless and that the dull abdominal pain had completely disappeared.

This case is illustrative of a severe dysmenorrhea with what Fontaine and Herrmann called a pelvic plexalgia. These cases are the type that should be selected for surgical intervention.

BLADDER DYSFUNCTION

Presacral neurectomy has been performed for the following vesical and ureterovesical disorders.

1. Learmonth, McConnell, Quinby and Douglass have reported improvement following presacral section for interstitial cystitis, however, not all of the patients were completely relieved. As the parasympathetic fibers carry afferent impulses from the mucosa of the bladder, presacral neurectomy can relieve only painful impulses of muscular origin, those of spasticity of the internal sphincter and whatever distress may be alleviated by ischemia of the vesical wall. In addition, an allied condition, irritable bladder without demonstrable cause, has been relieved by Braasch and Craig when this operation was utilized.

2. Some degree of relief in painful condition of the bladder as a result of malignancy has been reported by Learmonth and McConnell. This condition is seen in a debilitated patient with a short tenure on life, and it is my impression that the operation is too drastic. Also, little hope for complete relief from pain can be held because of the extent of the growth and transmission of painful afferent impulses over a larger distribution than that of the presacral nerve. I have found that intraspinal alcohol injection has been far more satisfactory with a minimum of surgical risk in this condition.

3. Ward has reported improvement following presacral neurectomy in hydroureter. Before submitting a patient to this procedure, one should determine definitely that this condition is caused by a spastic state of the ureterovesical orifices so that the operation will be on a sound physiologic basis. I have never resorted to presacral neurectomy in any of these instances.

4. Paralysis of the bladder may be approached with the thought that removal of the inhibitory sympathetic fibers may aid in the emptying of this organ. The results in cord bladder of non-syphilitic origin have been more favorable than those obtained in syphilitic patients. Learmonth, Bailey, Foulds, Calley and Fulcher have reported good results in paralysis of the bladder which followed trauma, congenital defect of the parasympathetic system, pressure on the cauda equina by tumors, fibrous, fibrocartilaginous or bony structures.

Cord bladder as a complication of syphilis has not responded as well, although Calley and Verbrugghen have met with partial success. In my experience, the case of syphilitic cord bladder will receive benefit from presacral neurectomy to the extent that the amount of residual urine will be less. Four cases of paralysis of the bladder have been subjected to this operation; two cases of syphilitic cord bladder received some benefit and the two other patients were cured. The following case reports are typical of the results obtained.

Case III. An unmarried man, 40 years of age, had contracted lues eight years ago and received very little treatment. Four years ago, he developed a tabes dorsalis with incontinence of the bladder. One year ago cystotomy had been performed for acute retention of urine. The wound healed with great difficulty and would occasionally break open. On April 12, 1934, resection of the presacral nerve was performed because of distress in the bladder region and a residual of 400 c.c. of urine in twelve hours. Following the operation, the patient had less discomfort, but still carried a residual of 120 c.c. per twenty-four hours. This condition has persisted to the present time.

Case IV. An unmarried woman, 20 years of age, complained of an inability to void or

defecate unless massive doses of castor oil were taken. The bladder and bowel would empty every four days and this was associated with severe pain. This condition had persisted for two years and was also associated with a severe dysmenorrhea. The past history was negative except that the menses did not appear until she was eighteen and that for two years she suffered from blanching, discoloration and pain in both feet when exposed to the cold or emotional stress. Examinations were negative except for an atomic bladder and colon, and the presence of Raynaud's disease of the lower extremities. The patient was not considered to be suffering from hysteria and the presence of Raynaud's disease seemed to indicate an autonomic imbalance. On October 21, 1932, a laparotomy was performed under anesthesia, and the presacral nerve and both lumbar sympathetic ganglia were removed. Immediately after operation, the lower extremities were warm, dry and pink. Four hours after the operation, the patient voided 150 c.c. of urine with no difficulty. It was necessary to administer enemas for seven days before a spontaneous bowel movement occurred with the aid of mineral oil. However, the patient was able to void three and four times daily after the operation. On the fifth post-operative day, the menses appeared with no distress. Since dismissal from the hospital, the patient has been able to void three and four times daily, and there has been a daily bowel movement without pain. The feet have remained warm and pink, and the menses have been painless.

This above case is illustrative of the relief obtained in an imbalance of the neurogenic apparatus of the pelvic organs. It is obvious that such cases will receive distinct benefit from sympathectomy if careful discrimination is used in selecting each case.

BOWEL DYSFUNCTION

There are two conditions of bowel disorder which are amenable to resection of the presacral nerve.

Gigantism of the bowel or Hirschsprung's or Mya's disease is best described in Hirschsprung's report in 1866 as "a condition of congenital high grade dilatation of the colon with thickening of all its tunics, especially the tunica muscularis, with retention of large quantities of fecal matter." Although this condition bears Hirschsprung's name, Barrington-Ward credits Ruysch with having reported a case in the seventeenth century; whereas, Billard, in 1820, Parry in 1825, and Bright in 1837, are believed to have reported earlier cases. Lewitt, in 1867, reported the first case in the United States and Finney's survey revealed that up to 1908, 206 cases had been collected.

The etiologic factor in this condition is not known and there is a multiplicity of theories. Rankin and Learmonth list these hypotheses under five general headings: (1) congenital defects, such as cause Hirschsprung's or Mya's disease; (2) obstructive processes, such as elongation of the mesentery, torsion of a segment, or multiplication of the intestinal loops; (3) anatomic factors, such as valve conditions, aplasia of the musculature immediately above the rectum, mechanical obstruction, and general systemic conditions; (4) infective processes, and (5) neurogenic processes.

The fact that certain cases of megacolon were relieved by lumbar sympathectomy has pointed to the fact that some cases are the result of a derangement of the sympathetic nervous system. Rankin and Learmonth subscribe to the belief that there is a mixed pathogenesis so that this can best be explained by several factors, such as an embryonal defect which may be either in the motor mechanism of the bowel itself, or an ectasia of the intestinal wall, some mechanical, congenital defect which, although not obstructive, produces the three cardinal anatomic features of colonic gigantism; namely, dilatation, elongation and hypertrophy. No changes in the resected nerves have been demonstrated. It is the general opinion that many of the changes in the bowel are secondary to some unexplainable type of obstruction low in the colon, intrinsic, neurogenic or mechanical.

The gross pathologic changes are those of thickening of all the layers of the colon,

except the serosa in which there is little change. Histologically, there is a thickening of the submucosa and mucosa, an hypertrophy of the circular and longitudinal muscular fibers. Lymphatic channels are increased and there is also an enlargement of the lymph nodes.

The symptoms of megacolon are most pronounced early in childhood, although some cases have been reported in the eightieth year of life. Males are afflicted three times as often as females and the symptoms are usually noted immediately after birth. Those cases appearing in adult life have a history of obstipation which was unrelieved by enemas and cathartics over a long period of time. The failure of the bowels to move, the abdominal distention, the flaring out of the lower ribs and the glazed skin are the most classical symptoms. Occasionally, the constipation is associated with intermittent diarrhea and yet the condition seldom progresses to actual obstruction and vomiting. Toxic symptoms are manifested by emaciation, general malaise, dry skin, dull feces and obtund mentality. Long standing cases are marked by a characteristic facies. The differential diagnosis need only consider the history, a general and roentgen examination and elimination of rickets, tuberculous peritonitis, ovarian cyst and chronic ileus.

Resort to various surgical procedures have been done in this condition. Some of the old methods, such as resection or side-tracking, bore a high mortality and poor results.

Wade and Royle achieved considerable success with a left lumbar sympathectomy, as did Robertson. Later Judd and Adson found bilateral lumbar sympathetic ganglionectomy to be very effective. Later Rankin and Learmonth resected the inferior mesenteric nerve and the presacral nerve in moderately advanced cases with beneficial results. Adson is of the opinion that the degree of megacolon should determine the extent of sympathetic resection and only the cases in which the obstructive process is in the lower sigmoid or rectum will benefit by presacral neurectomy. I have had but one case of megacolon which seemed to justify sympathectomy.

Case v. A young, unmarried woman, twenty years of age, complained of obstipation to the extent that the bowels would move only every five or six days after drastic cathartics and enemas were administered. The patient was five feet tall, weighed 85 pounds and presented the characteristic picture of dull facies and distended abdomen. Because of the dull mentality, it was decided that novocain injection of the lumbar sympathetic ganglia should be performed. The patient was placed under the fluoroscope and it was noticed that four quarts of barium solution were required to fill the colon. On January 21, 1931, a bilateral injection of the lumbar sympathetic ganglia was carried out and the patient again placed under the fluoroscope where it was noted that less than one quart of barium solution was required to fill the colon. A lumbar sympathetic ganglionectomy was advised but the following day a spontaneous bowel movement occurred and since then the bowels have moved normally.

This unusual reaction is similar to Flothow's experience with a permanent relief from dysmenorrhea following novocain injection. Apparently, some chemical change took place in the sympathetic fibers which produced a stability of the neurogenic mechanism.

The other bowel condition which is amenable to presacral neurectomy is obstipation of rectal origin. In a small group of patients, there is a lack of expulsive power which appears to be on a neurogenic basis. However, there is no type of case in which deliberate judgment is so essential as in the individual with a faulty nervous mechanism who suffers from constipation. It is a well known fact that many neurotic patients unduly center their attention upon the colon and its function. The following cases will illustrate the pitfalls of a proper selection of cases for surgery.

Case vi. An unmarried woman, 28 years of age, was first seen in January, 1933, because of a right Bell's palsy, general nervous exhaustion and chronic constipation. No focus of infection

could be detected and means were instituted to placate the nervous instability and the patient was placed upon a rigid anti-constipation regimen. This patient also stated that each menstrual period was associated with moderately severe pain for four days. In three months, the Bell's palsy had cleared, but the constipation remained resistant to any form of treatment. Her past history had been negative except for peritonitis following a rupture of the appendix. It was feared that adhesions had formed which might have exerted a mechanical influence against the normal evacuation of the bowels, but roentgenograms of the intestinal tract were negative for evidence of pathology. On July 25, 1933, under avertin anesthesia, a laparotomy was performed and many dense adhesions of the pelvis were found. The sigmoid was kinked behind the left ovary and was densely adherent to the ovary and tube. A small cyst was found on the right ovary and this was punctured. The presacral nerve was then removed. The patient made an uneventful recovery and on the fifth postoperative day the menses appeared with no distress. Since the operation, the bowels have moved daily although it is necessary for the patient to take a small amount of mineral oil. Her general health has improved and she has been free from dysmenorrhea.

This case is rather difficult to classify and resection of the presacral nerve cannot be entirely responsible for the relief of constipation because of the mechanical influence which was demonstrated at operation.

Case VII. An unmarried woman, 34 years of age, was first seen in March, 1932, because of a bizarre facial neuralgia, multiple complaints, nervous instability and chronic constipation. Her past history was negative except for many nervous complaints which she attributed to family and financial burdens. Examination was negative and she was placed upon an anti-constipation regimen which was supplemented with efforts to help her regain her nervous stability. In December, 1933, this patient had much difficulty with constipation and as drastic cathartics had failed to relieve her, it was felt that resection of the presacral nerve would be justified. This was performed under avertin anesthesia on December 28, 1933. She made an uneventful recovery and until March the bowels had moved daily with no distress. However, since then it has been necessary to administer rather large doses of mineral oil to produce a daily evacuation.

In retrospect, it is my opinion that this patient has received only partial benefit from surgery. This case is indicative of the poor sufferer with a general instability of the central and sympathetic nervous systems and sounds a note of warning against operation unless of utmost necessity.

Case VIII. An unmarried male, 28 years of age, was first seen in February, 1934, because of headaches, vertigo, nervousness and chronic constipation. His past history was negative except for a tendency to assume the burdens of life too seriously and to be very introspective. He has suffered from severe constipation for years and would go as long as six days before a bowel movement would occur with the aid of drastic cathartics. Repeated enemas would often fail to empty the bowel satisfactorily. General and neurologic examinations, including spinal fluid, were negative except for the colon which was moderately dilated and emptied slowly. On April 25, 1934, under avertin anesthesia, the presacral nerve was resected and, with the exception of a few nervous upsets, recovery was uneventful. The bowels moved daily with no difficulty until August 1, 1934. At this time, the patient developed an acute retention of urine. The urologist's report revealed no evidence of a pathologic process and the patient was believed to be suffering from hysteria because of the necessity of assuming a few irksome duties. The bowels have occasionally failed to move for one day, but a small dose of mineral oil corrected any distress.

This case shows again the importance of evaluating the mental poise of the individual. Undoubtedly there has been some benefit from presacral resection in the last 2 cases, but there is a question as to the advisability of submitting this type of person to any operative procedure where only a small amount of inhibition can be removed.

SUMMARY

Eight patients with dysmenorrhea and seven with bladder and bowel dysfunction

have received benefit from presacral neurectomy.

The operation removes afferent pain fibers and the inhibitory elements which prevent emptying of the bladder and bowel.

Extreme caution in the selection of cases must be exerted lest a useful therapeutic agent be thrown into disrepute by untimely and ill-advised operations.

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INJECTION TREATMENT OF HERNIA*

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FOR the period of a little over one hundred years the cure of hernia by means of the injection method has been an idealistic dream of the medical profession. It has had its periods of agitation and experimentation; its ups and downs and final triumph as a recognized method in surgery.

The early history of this method characterized by its lack of knowledge, both of anesthetics and asepsis, as well as by its improper and inefficient technique and the use of solutions both painful and inefficient, resulted necessarily in its almost complete abandonment and a return to surgical treatment with the knife.

As time passed many new solutions were devised, new technique introduced, the use of local anesthesia and asepsis and antisepsis were used, until today in good hands not much is left to secure complete mastery over reducible hernia, except development of greater skill and manual and mechanical dexterity on the part of the operator.

Before one can qualify for the successful treatment of hernia by any method, it will be necessary for him to become thoroughly versed in the anatomy of the hernial area, as well as in the direct and predisposing causes of this affliction.

Examining the anterior abdominal wall from behind, it will be seen that there are five more or less distinct ridges by which are formed on each side three peritoneal fossae, known as the inguinal pouches (fovea inguinalis). These fossae, or grooves, act as a sort of funnel in which the intestines glide whenever intra-abdominal pressure is increased. These fossae or grooves are described as the external inguinal fossa (fovea inguinalis lateralis) which lies outside the deep epigastric artery, and leads directly to the internal abdominal ring; at its lower point is a dimple or depression of the peritoneum corresponding to the point from which the processus vaginalis passed down with the descent of the testis.

The middle inguinal fossa (fovea inguinalis mesialis) lies between the deep epigastric and the obliterated hypogastric arteries. The internal inguinal fossa (fovea inguinalis interna) lies internal to the obliterated hypogastric artery, that is between it externally and the urachus internally. Thus, it will be seen that the middle fossa corresponds to the outer division of Hesselbach's triangle and the inner fossa to the inner division.

Another fossa of the peritoneum is seen near this region and it lies just beneath the inner part of Poupart's ligament; this is known as the femoral fossa (fovea femoralis). This fossa leads into and is in fact really a guide to the femoral canal.

Leaving the inner abdomen, let us take a brief review of the hernial area of the abdominal wall. This contains three points of interest; because of the frequent weakness of the wall at these points. (1) The internal abdominal ring, located about midway in the length of Poupart's ligament and a half to three-quarters of an inch above this ligament; in many cases this ring is abnormally open and contains but little fibrous tissue with which Nature should have closed it and when therefore any increase in intra-abdominal pressure occurs either from sudden strains, as from coughing, sneezing, straining at stool, heavy lifting, jumping, this weakness is augmented.

Or (2) increased abdominal pressure of a long continued nature may occur as from accumulating fat in the omentum, mesentery and peritoneal tissues. This accumula-

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tion lessens the size of the peritoneal cavity and hence increases the pressure.

(3) A lengthened mesentery may be a factor in hernia by allowing the intestine to sag more than normal and long continued feeble health may result in muscular relaxation of the abdominal walls leading to enteroptosis, which is quite likely to at last lead to a further enlarging of the abdominal ring before it is finally stretched open by a hernial descent.

Direct hernia would be as common as indirect hernia, were it not for the fact that the transversalis fascia is normally a strong backing for Hesselbach's triangle. However, a weakness or break in the continuity of this fascia sometimes occurs and then sliding down the middle or internal fossa the intestine breaks through and a direct hernia is the result: in similar manner with a defective supply of fibrous tissue in the femoral canal leaving an open femoral ring, the intestine comes sliding down the femoral fossa, pushes a sac of peritoneum before it and a femoral hernia appears.

Any hernia may seem to appear suddenly but the fact is in most cases the causes have been long at work and may generally be said to have existed from birth. In short, the predisposing causes of hernia are plainly vestigial weaknesses left as a result of evolutionary development in prenatal life. As an interesting biological fact in this connection may be mentioned that abdominal hernia among monkeys and apes is almost unknown, despite the fact of their jumping and leaping through the trees and numerous severe strains received in the activities of their arboreal life.

Perhaps it may be that man in his superior mentality in the process of evolution, became more ambitious to maintain the upright posture than his primate brother and thus pays the penalty of his upright position, from which the primate escapes because of still maintaining his method of travelling on all fours. Be this as it may, the fact remains that a lengthened mesentery, a gastroenteroptosis, the presence of the inguinal fossa and increased intra-abdominal pressure are plainly not to any great extent remedial by means of the surgery of today and therefore, except as abdominal fat, should receive some attention from a dietetic standpoint, there remains but one effective means for the prevention and cure of inguinal and other forms of abdominal hernia, and that is to so repair the weak spots in the abdominal wall as to make a hernia impossible, despite the prenatal predisposing factors.

To the many millions of sufferers in the United States from unrelieved hernias, any advance in the method of operating on or treating hernia that will offer greater safety and efficiency in the curing of this affliction will appear as a godsend.

To the surgeon who has been compelled to refuse relief to the thousands who from chronic bronchitis, bronchiectasis, cardiac disease, weaknesses of age, etc., any method that will place these cases in the curable class will be hailed as a welcome and timely discovery, for it is the general practice of nearly all surgeons to tell all these classes and particularly the aged, to avoid the operation for the relief of hernia so long as a truss can be satisfactorily worn. Because of this and for many other reasons there is sometimes great difficulty in getting consent to a really necessary operation.

Industrial surgeons have many cases of hernia coming into their hands, in which the hernia is claimed as a result of an injury received while on duty and the element of a claim for damages from personal injury in such cases is always present.

For this reason and for many other reasons as well, if one can hold out the certainty of a radical cure for the rupture by the injection method without the risk of an operation, and with little discomfort and no hospitalization and no time lost from work, the handling of such cases may be made much more simple and the expense to the employing company much less. Already many of the large railway companies are adopting the method. Many insurance companies are interesting them-

selves in this ambulant treatment and it is only a question of a short time before practically all companies will adopt it.

A hernia may be defined as the protrusion of a viscus or viscera through a natural or accidentally produced opening in one of the walls of the body. A rupture, strictly speaking, is the opening through which the viscus or viscera pass.

The coverings of a hernia include the skin and in their order all the integuments which underlie it. These coverings vary according to the locality of the hernia.

The hernias with which we most frequently have to deal are the indirect inguinal, direct inguinal, femoral and umbilical; the many other forms of hernia which occasionally occur are not encountered often enough to warrant attention in this paper.

It is not the object of this article to discourage surgery in the handling of hernias but to call attention to the fact that in every case of reducible hernia in which the propriety and safety of the radical operation is questionable we have an alternative method which is as effective.

The injection method is practically free from danger and not at all confining, is entirely ambulatory; the individual is not required to stop work, which is of course an economic gain to himself and his employer and therefore is naturally a preferred procedure.

Experiments upon dogs and other animals show that with the use of mildly irritating injections into the fascia and muscles of tannate and gallate solutions of different plant principles, within a few hours there is a definite reaction manifested largely by edema of the tissues and a production of exudate of plasma. Later, this reaction is replaced by the production of fibroblasts; these fibroblasts later appear as young fibrous tissue cells and later these cells develop into mature fibrous tissue.

At the end of two weeks these cells appear well developed, and for each injection, there is an area about 2 cm. in diameter of fine fibrous tissue.

If one is able to produce good strong, healthy fibrous tissue *where he wants it*, it is logical that injections into the inguinal canal and around the rings will accomplish the same results as a successful operation, for surgery does nothing more than close the rings and canal except to remove the sac, which is destroyed equally as effectively by fibrous union.

The first essential requirement for success in the injection treatment of reducible hernia, is a comfortable, well fitting truss, that will keep the hernia in place under all conditions, while the treatment is in progress. It is not sufficient that the patient says that his truss is all right, it is the duty of the physician to see for himself, and to be able to select and adjust trusses under all conditions and for all forms of hernia. No one make or style of truss is appropriate for all cases, but in general a steel spring type truss will prove better for the day, while a web elastic with a springy pad is more comfortable and sufficiently effective for the night. A truss should be worn both day and night during the treatment and the truss should always be removed or applied *while lying on the back.*

No rules for fitting trusses can be formulated that will not have some exceptions, but I will say that the ordinary single pad, one-sided spring truss will be suitable for many or most inguinal hernias, and when fitted with a round, convex, springy pad, will do for many direct inguinal hernias. The frame truss is the most comfortable and effective in double, direct or indirect hernias, except where one's work requires lying flat on the back as in working under an automobile; in these cases the truss is raised up clear from the body when in the supine position. In such cases an elastic web truss *tightly drawn*, is sometimes better for the day and it may be somewhat loosened for night wear.

A cross body truss with stiff spring and round springy convex pad is generally more effective for a very fat man with a single hernia especially of the direct

variety, and this is also generally better for a femoral hernia, although the pad should be small. The umbilical truss should be a spring truss open in the back, that is, a frame truss with a central pad in front.

Before commencing treatment the whole hernial area should be explored thoroughly; a careful differential diagnosis should be made. The cord, the rings, the canal should be examined; all malformations or complications should be noted for very obvious reasons.

The general health should be investigated and any patient who gives evidence of tuberculosis in a late stage, of diabetes of a hopeless character, or one who is afflicted with a severe asthma of a steadily confining character, or an individual with advanced nephritis should be refused treatment. Diabetes is not a contra-indication to treatment except in advanced cases.

One should be absolutely certain that a hernia is completely reducible, and, of all things, an omentum that does not completely go back should not be overlooked, for an accidental injection of an incompletely reduced omentum would be quite likely to form adhesions to other hernial contents or to the inguinal canal, and, in either case completely prevent a cure and render an operation practically imperative.

Having decided that a hernia is reducible and the patient a suitable case for the injection treatment, the procedure for treating an indirect inguinal hernia should be as follows: Put the patient on his back on the office operating table, clip the hair from the hernial area over a broad surface. Wash with alcohol after a thorough sterilization of the skin.

Put out two sterile glass syringes, preferably of 5 c.c. capacity. In one syringe place 2.5 c.c. of 2 per cent procaine solution and in the other syringe 3 c.c. of the proliferating solution. The procaine loaded syringe should have a 22 gauge needle of sufficient length to penetrate the fascia of the external oblique muscle. In an average individual this will be about an 1½ inch needle. In a fat person the needle should be 2 inches or even 2½ inches and sometimes, but rarely, 3 inches long.

It must be an exceedingly spare individual who will require an inch needle or less, yet such cases do occur. Your needle must penetrate the fascia. It must not enter the abdomen; it must pass through the fat, as the injection can do no good in the fat tissue and will prove unnecessarily irritating.

My own technique to get the depth, I have worked out very carefully, and if the operator will follow this plan, he will find it simplicity and certainty itself. In each case the first thing to do before injecting the fluid, is to locate the internal ring, which will be found midway of the length of Poupart's ligament and about 1 cm. or 1.5 cm. above this ligament. Before proceeding further, place the patient in a moderate Trendelenburg position.

Select a needle that is supposedly long enough to penetrate the external oblique fascia; plunge the needle quickly through the skin at just below and inwardly from the upper edge of the internal ring; now slow up immediately after penetrating the skin and go slowly through the fat. The needle will go through the fat as though passing through butter, and in this way, the slightest change in resistance may be observed, and even the sharp point of a long bevel needle will be distinctly felt to strike and penetrate the fascia by one who has the classical tactus eruditus; the needle will be felt to pass through the fascia. Personally I do not believe that the needle should be allowed to pass much farther through the fascia than just to give a distinct clearance for the eye of the needle. If the operator has not the requisite skilled delicacy of touch he would better use the short bevel needle, as this can be more distinctly felt to touch the fascia, until skill has been acquired.

After penetrating the fascia to the required depth, withdraw the piston slightly to see if any blood arises into the syringe. If no blood is seen to enter the

syringe, inject 2 c.c. of the procaine solution *very slowly*. Then uncouple the syringe and leave the needle in situ, laying a little sterile gauze over it. After an interval of ten minutes couple onto the needle thus left in situ, the syringe loaded with 3 c.c. of proliferating fluid and inject slowly until the 3 c.c. of proliferating fluid has been introduced. This operation should be accomplished so slowly as to seem like no movement of the piston can be readily seen, or at about the rate of 1 cm. per minute.

Again uncouple the syringe and attach the procaine syringe with its 0.5 c.c. of procaine and inject, in order to wash out the proliferating fluid from the needle to prevent the back flow of the proliferating fluid through the withdrawal of the needle; then take a wisp of cotton, (sterile) and have the patient press moderately over the needle prick until the fluid has an opportunity to infiltrate the tissues, after which the needle prick should be touched with a little iodine, mercurochrome, bismuth violet or any other favorite antiseptic you may please.

Have your patient lie quietly on the table about fifteen minutes and then after placing a small dot of adhesive plaster over the needle prick as a marker, put on his truss, well fitted, and let him go about his business until the next treatment, which is best given in about two or three days.

The next treatment should be given in the same manner as the first and I would suggest that the next four after the first, as a general rule, should be placed around the internal ring, above, below and to the right and left from the ring, after which the following injections should be placed just above and mesial to the cord, at intervals of distance of about one-third of an inch until the external ring is reached and then an injection should be made into the lower part of the conjoined tendon, one a trifle higher up and finally one more just missing the cord, directly into the external ring. Ordinarily this will complete the treatment of an indirect inguinal hernia.

The treatment of a direct inguinal hernia differs from that of an indirect inguinal hernia because the injections are not made into the external oblique fascia but into the conjoined tendon and the Transversalis fascia as well as into the external ring.

My technique for a direct hernia is usually about as follows: First, I place an injection into the conjoined tendon low down, then one higher up and so on until I reach the level of the upper part of the hernial opening; next comes an injection deep into the tissues just above the hernia, after which the entire Hesselbach's triangle is to be reenforced and finally injections directly into the external ring avoiding the cord.

A direct inguinal hernia is often very difficult to keep in place, but in the hands of an expert it may be cured by a less number of injections than the average indirect inguinal.

Personally, I would not advise one to undertake the treatment of femoral hernia by this method until he has familiarized himself by the successful treatment of both varieties of inguinal hernia, because of the greater difficulties and dangers in handling femoral hernias.

However, the following method of treatment of a femoral hernia, which I have worked out will simplify and render safe even the treatment of femoral hernias. With the patient in a marked Trendelenburg position, I make sure of the entire replacement of the hernia and sac. I then find the pulsation of the femoral artery, and remembering that the femoral vein lies to the inner side of the artery, I place my index finger of the left hand over the vein, with its palmar surface against the artery, and then introduce an inch needle into the femoral canal internal to the index finger of the left hand. By doing this neither the vein nor the artery will be injured. Leaving the patient in this Trendelenburg position after the proliferating solution is introduced, I make a compress of surgeon's cotton saturated with flexible collodion and press this up against the femoral ring, and

after this has begun to set, I place the truss pad over it and let the patient continue about his work. It will be found, when he returns, that the hernia has kept its place. From seven to nine injections are usually sufficient to cure a femoral hernia. I have sometimes found it useful when treating a bad femoral hernia to place two or three injections to the mesial side of the femoral vein and artery just where they pass under Poupart's ligament to enter the thigh and sometimes even down just behind the horizontal ramus of the pubis at the point where the artery and vein cross the bone to the mesial side. This is also sometimes a good practice in inguinal hernias where a weakness is evident at this point; this is not infrequent in gastroenteroptosis subjects. In doing this be sure that the patient is in an exaggerated Trendelenburg position.

The umbilical hernia of infants should not be treated by this method, because nearly all of them will disappear before the age of 3 years, by the widening and development of the recti abdominalis muscles, if the mother is taught how to keep the hernia retained by a broad pad fastened with adhesive tape and the proper instructions that go with it. In older children and adults the treatment consists in using the frame truss with central pad for the day and an elastic truss with pad at night. Injections should be given directly into the edge of the muscle around the hernia, remembering that the muscle is rather thin at this point and to go too deep will cause a severe belly ache, though no permanent harm will result.

The cure of properly selected navel hernias is easy, except the very large ones, which should not be undertaken. These large ones are best treated by the Mayo operation or some modification thereof.

Infantile hernias in the inguinal region are generally best left until a child is old enough to wear a satisfactory spring truss. After the age of eight or ten years one may reason with the average child and local anesthetic may be used. To attempt this in a young child invites violent struggles which are harmful and unnecessary and the use of a general anesthetic as many times as necessary for the cure of hernia is inadvisable in the experience of most physicians.

And now I shall consider a question most frequently asked in connection with the ambulant treatment of hernia: "What solution do you use?" There lies before you a choice of twenty solutions more or less used for this purpose.

It is true that microscopic examination of the tissues formed by the reactions of these various solutions shows that there is very little difference in quality or character of the tissue formed but this does not tell the whole story. The many solutions containing phenol, zinc sulphate and other intensely irritating chemicals must necessarily be given in quantities so small as to result in very small nodules of fibrous tissue and hence cannot produce the smoothly continuous interlacing sheets of new tissue which result from the use of mild irritants, which can be given in quantities of from 3 c.c. to 5 c.c. at an injection resulting in broad interlacing areas of new tissue; the results are to be roughly compared to a stitch of repair here and there on the one hand to a vulcanizing of the weakened area into one strong mass, as suggested very aptly by Puleston.

Further, we must consider the idiosyncrasy of many people which would necessarily bar the use of phenol in many cases. I am perfectly willing here to recognize the remarkable results attained by Ignatz Mayer with his solution which contained both zinc sulphate and phenol but Mayer was a very skillful man who gave treatment at long intervals and with great care which gave results, few if any others, could have obtained, unless used as he did, by keeping a patient from twelve to eighteen months under treatment.

And now this brings us to the consideration of a class of agents containing only tannate and gallate principles of harmless plants, which may be freely used in quantities running from 3 c.c. to 5 c.c.

Pina-Mestre hernial solution belongs to this class and has been fully tested in its present form over a period of fifteen years and the patients first cured still stand the test of time. Many other solutions of alleged similar content have recently been introduced and you may wish to try them. These preparations may be safe and I do not know of any reason why you should not use them, but I personally feel that the five year test should be applied to all like agents. Time will give each preparation its proper place.

I have never known a failure in the use of the preparation which I use (Pina-Mestre) that could by any stretch of the imagination be charged to the medicine.

SUMMARY

The early history of the injection treatment of hernia was marred by unsafe solutions, lack of local anesthesia, faulty technique; time corrected all this and made the method one of the modern triumphs of medical progress.

The essentials of success are a mastery of the causes of hernia; an exact anatomical knowledge, a close discrimination between the surgical and nonsurgical indications, a thorough training in the art of truss-fitting, and co-operation on the part of the patient.

Direct and indirect inguinal, femoral and umbilical hernias come within the scope of this method, except occasional contraindications. The reducibility of a hernia and possibility of keeping it reduced, rather than size will control success. The mastery of technique is absolutely essential to success, as well as an efficient and safe solution. No powerful sclerosing solution should be used.

FRACTURES: General Principles of Treatment. The first aim of treatment is to save life, which may be endangered by shock, inflammation, pneumonia, or bed-sores. The second aim is to save the injured limb. The third aim is to obtain as perfect reduction and restoration of function as possible.

From—"Elements of Orthopaedic Surgery" by N. Ross Smith, M.B.

INJECTION TREATMENT OF HERNIA:

A CRITICAL ANALYSIS OF THE FAILURES, RECURRENCES AND COMPLICATIONS*

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WITHIN the past few years a reinvestigation of the subject of the injection treatment of hernia has resulted in the establishment of the fundamental principles upon which this therapeutic measure is based. Experimental work on the various solutions used in this treatment show, beyond a doubt, that these solutions are efficacious in producing fibrous tissue.[7] The mechanism of hernia and the rationale of the treatment have been well established.[8] The mechanics of trusses and the importance of trusses in this method have been pointed out. Numerous investigators have proved the feasibility of this method, and have reported cures in many hundreds of cases. It is important now to analyze critically our results to date, to evaluate statistically the good results, to point out the percentage of failures, to discuss the complications, and to seek the underlying causes of such failures and complications as occur with this procedure.

Before attempting to analyze critically the results of this method of treatment it is advisable to point out briefly the proved principles on which this method is based, and to state in detail the present outline of treatment. The fundamental causes of recurrence and failure with this method depend to a large extent upon the neglect of one or more of these principles.

The essential factors necessary for a complete understanding of this method may be grouped under the following divisions: (1) the anatomical basis, (2) the correct differential diagnosis between direct and indirect hernia, (3) the choice of solution for the injection, (4) the choice and application of the proper truss, (5) the technique of injection, (6) the placement of the injections, and (7) the outline of treatment and follow-up. A thorough knowledge of these principles, and familiarity with their application are necessary for a critical analysis of this method.

1. *The Anatomical Basis.* The hernial sac, in the indirect type of inguinal hernia, enters the inguinal canal at the internal ring, and passes downwards in the canal beneath the transversalis fascia and internal oblique muscle. In the canal's medial third it emerges from beneath the lower edge of the internal oblique muscle, and lies then deep to the external oblique muscle. An approximation of these muscular layers prevents the distention of the sac by its contents. This result may be obtained by the injection of a solution which produces fibrosis between the muscle layers which obliterates the patency of the inguinal canal. Similarly, in a direct hernia, approximation of the muscles and fascia in Hesselbach's triangle will accomplish the same result.

2. *The Differential Diagnosis between Direct and Indirect Hernia.* The importance of this differentiation cannot be overemphasized, for on it depends the placement of the injections and the placement of the truss. As can be seen from the anatomical considerations cited, in a direct hernia the sac emerges from the abdomen through Hesselbach's triangle, and the position of the injections, as well as the placement of the truss, must be over an area considerably removed from the internal ring. This subject has been considered in detail in another communication.[6]

* From the Department of Surgery, Mount Zion Hospital, Service of Dr. Harold Brunn.

3. *The Solution for the Injection.* Two principal types of solution recommended for this procedure are alcoholic solutions

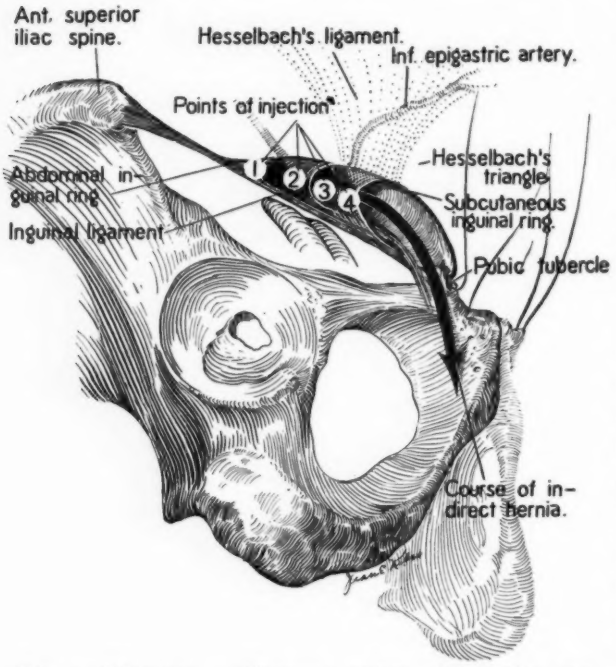

Fig. 1. Diagram to illustrate points of injection for an indirect inguinal hernia. Two to four injections are given at each of the figured points on the diagram. Note that the first injection is over the internal ring, and that subsequent injections are given down the canal toward the external ring.

of tannic acid and solutions containing the salts of fatty acids. These solutions are relatively non-irritating, do not produce a severe inflammatory reaction and are non-injurious to tissue. They have been experimentally proved to produce fibrous tissue and to be nontoxic. The necessity of experimentally proving the efficacy of solutions to be used in this treatment cannot be stressed too much. The indiscriminate use of unproved solutions will result in danger to the patient and failure of the treatment.

4. *The Choice and Application of the Proper Truss.* Increasing experience with this method of treatment has emphasized the necessity for a better understanding of this phase of the procedure. This subject is discussed in detail in a previous paper.[6]

5. *The Technique of Injection.* Complete familiarity with the technique of this procedure must of necessity form a major part of the method. The sterilization of the instruments, the choice of the needle and the technique of insertion of the needle determine largely the dangers and results of the treatment.

6. *The Placement of Injections.* In a previous communication a technique was described which, in an indirect inguinal hernia, required starting the injections at the external ring and gradually working up the canal.[5] While the results of this method were good, it was felt that the technique of injection could be simplified by starting the injections at the internal ring, and placing the subsequent injections down the canal, with the last injection over the external ring (Fig. 1). With this latter method the lowermost portion of the canal is left free of swelling and induration until toward the completion of the treatment. This makes the location of the points of injection somewhat easier and gives the operator a better guide to the placement of subsequent injections. It has the further advantage of closing the uppermost part of the canal first. This portion of the canal is probably the most important to close, since any opening in this area prevents cure of the hernia. Further experience may show that the ultimate results are identical whether the injections are begun at either end of the canal. Further experimental work seems to prove so far that fluid injected into the inguinal canal at any point tends to disseminate equally throughout the canal.

In the earlier cases injections were begun at the external ring since it was felt that any unexpected descent of the sac contents would merely descend to the area of fibrosis. If injections were begun at the internal ring first, any untoward descent of bowel might result in a strangulation at the point of a narrowed but incompletely closed internal ring. However, an increasing understanding of the mechanics and application of trusses, and the use of a proper type of truss, which would maintain closure of the internal ring, have obviated this danger.

7. *Outline of Treatment.* The procedure, which is used at the present time and which we feel will give the least number of recurrences, is as follows: The first injection is placed over the internal ring, as is likewise the next three or four. This is usually sufficient to close the internal ring completely. The subsequent injections are placed consecutively down the canal until the external ring is closed. At any given point several injections may be given to fill the inguinal canal completely with fibrous tissue over that area. The total number of injections for this phase of the treatment should be at least eight and as high as twelve. The injections are given three times weekly on alternate days. They are continued until the hernia is closed and does not protrude when the patient coughs or strains in the flat position.

During the subsequent four weeks, injections are given once a week over the internal ring, or at other areas down the canal, if according to the judgment of the operator such areas show any weakness of the fibrous wall. These injections are for the purpose of reinforcing the treated area and are of importance when recurrences are considered. This "over-treatment" is necessary due to the amount of tissue resorption that occurs, the amount varying with the individual and is often greater in older patients. Whether this is due primarily or only incidentally to age remains as yet undetermined. In such cases it may be advisable to prolong the

HERNIA CASE REPORT—MOUNT ZION HOSPITAL

NAME.....

SERIES No.....

HERNIA No.....

DIAGNOSIS.....

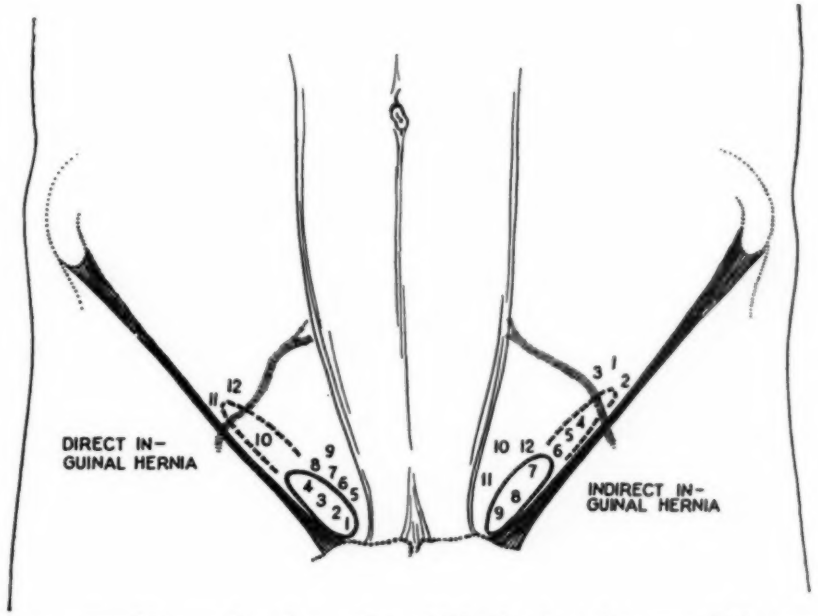

Fig. 2. Graphic record to show points of injection in a given case. This type of record eliminates the necessity of indicating points of previous injection by means of adhesive tape or tattoo marks on the skin.

reinforcing phase of the treatment to eight or ten injections, given at weekly intervals. A graphic chart is kept to show the points of injection (Fig. 2).

During the next six months the patient should be examined once monthly. The truss, which is removed at night one month following the last reinforcing injection, must be worn during the day. During this period of immediate follow-up, careful examination should be made at each visit for evidence of weakness or recurrence. If this should happen, careful search for the cause must be sought and appropriate measures taken for its correction.

The period of final follow-up extends over the subsequent year. During this period the patient is examined every two months. If at the completion of this time the hernia remains closed, without evidence of recurrence, the patient is discharged as cured. Table I presents an outline of a complete course of treatment.

TABLE I
OUTLINE OF COMPLETE COURSE OF TREATMENT OF HERNIA BY THE INJECTION METHOD

Phase	Time	Description
1. Preliminary treatment	1 month	8 to 12 injections, 3 times a week, until hernia is closed.
2. Reinforcing treatment	1 month	4 injections, once a week.
3. Immediate follow-up	6 months	Examination once a month, for 6 months. If necessary, occasional reinforcing injections.
4. Final follow-up	1 year	Examination every 2 months, for 1 year.

Note that the total length of treatment requires twenty months for observation of each case.

Neglect of these fundamental principles will result in failures and recurrences. Following an analysis of the cases in our series the reasons for recurrences will be discussed in detail.

RESULTS OF INJECTION METHOD TO SEPTEMBER, 1936

Since the inception of this work early in 1934, 225 cases of reducible direct and indirect inguinal hernias have been treated by the injection method in the Hernia Clinic, Surgical Division of the Out-Patient Department, Mount Zion Hospital. The total number of injections given has been over 4500. Although it is too early to state definitely that the results are equal or better to those obtained by the recognized surgical procedures, we can say that up to the present time the results have been sufficiently satisfactory to warrant continuance of this work. For purposes of statistical analysis the cases have been divided into two groups. These have been designated as, (1) the operative group, or that group which includes cases that would ordinarily be classed as good operative risks; and (2) a non-operative group, including such patients who for some reason are poor surgical risks. In the first group our statistics show that the number of recurrences and the ease of cure, compare most favorably with operative statistics. In this group there have been a total of 168 patients. The treatment in 121 is complete, and the follow-up on these cases has been from six to eighteen months. There have been five recurrences, giving a recurrence rate of 4.1 per cent. In the second group there have been fifty-seven patients, treatment being completed in forty-six. It may be stated at this point that the cases in this group were denied surgery and that the only treatment formerly accorded this type of patient was the application of a truss. For such reasons as age, complicating cardiac or other medical conditions these cases would never be operated upon. The cures which result in this group show a definite advance in the treatment of hernias. Surgery could offer these patients no hope of relief. In forty-six patients there have been nine recurrences, an incidence of 19.5 per cent. Tables II and III summarize

the results of injections and the incidence of recurrences.

Table II
RESULTS OF INJECTION OF HERNIA TO SEPTEMBER, 1936

	Completed Cases	Cases under Treatment	Total
Inguinal—good surgical risk.. (Direct and indirect) Average age 37 years	121	47	168
Inguinal—poor surgical risk... (Direct and indirect) Average age 68 years	46	11	57
Total number of cases...	...	..	225

Table III
RECURRENCES
All cases with 6 to 18 months follow-up after completion of treatment

	Inguinal Good Surgical Risk	Inguinal Poor Surgical Risk
Number of completed cases.....	121	46
Number of recurrences.....	5	9
Percentage of recurrences.....	4.1	19.5

A consideration of the reasons for the recurrences, 14 in all, in both these groups of patients showed deviations from the fundamental principles outlined. With corrections made as necessary for the individual case these cases are being retreated. All but two are completely closed and under treatment at the present time.

FACTORS WHICH INFLUENCE RECURRENCES, FAILURES AND COMPLICATIONS

With an understanding of the fundamental principles of the injection treatment of hernia in mind the factors which tend to produce recurrences, failures and complications may be considered.

Anatomical Basis and Diagnosis. Perhaps the most frequent cause of recurrence and failure in the injection treatment of hernia is the fact that an incorrect diagnosis is made previous to the start of the injections. As can be seen from the consideration of the anatomical relationships of the inguinal region, there is a considerable difference in the muscle and fascial arrangement in a direct and indirect hernia. An incorrectly diagnosed case will be incorrectly treated. If a diagnosis of indirect inguinal hernia is made and injections given over the inguinal canal, in the case of a direct hernia, no cure will result. In a direct hernia the injection must be over Hesselbach's triangle. Similarly, if injections are given over Hesselbach's triangle in an indirect hernia, no cure will result. The frequency of direct hernias has been, we feel, considerably underestimated. Many cases of so-called "saddle-bag" sac are overlooked. Such an oversight would result in a persistence of the direct element of the hernia following therapy and an incorrect assumption of failure of the method would result. In our series of cases there has been 31 per cent of direct hernias, that is either simple direct, recurrent postoperative direct, or direct associated with an indirect hernia.

To overcome the possibilities of failure or recurrence due to these factors, special care should be given to the diagnosis. In order to obviate the oversight of either a direct or indirect element in any given case it may be advisable to give three or four injections over Hesselbach's triangle, in the case of an indirect hernia, following the completion of treatment; and similarly to give three or four injections over the internal ring, in the case of a direct hernia. However, accurate diagnosis will make this latter more or less haphazard procedure unnecessary.

Failure of the treatment and serious complications will follow the application of this method to any irreducible, incarcerated, strangulated or sliding hernia. The failure will be due to the fact that the contents of the sac prevent the approximation of the muscles and fascia in the canal. The complication may be perforation of the bowel, gangrene of the bowel,

peritonitis and fecal fistula, and will be due either to perforation of the incarcerated sac contents by the needle or pressure from the truss pad. The treatment of a hernia associated with an undescended testis will likewise result in failure of cure of the hernia and further damage to the testis. All of these conditions must be treated by operative means.

Solution for the Injection. As stated, a fundamental principle in this procedure is the use of proved solutions, by which the demonstration of four important factors is meant: (1) the production of fibrous tissue when injected into the muscles of experimental animals; (2) the lack of toxicity when injected into the peritoneal cavities of experimental animals; (3) its sterility when tested by bacteriological procedures; and (4) the cure of hernia cases in carefully followed clinical series. The violation of any of these four factors in the use of unproved solutions will result disastrously. Any solution which fails to produce fibrous tissue will necessarily fail to close hernias. A solution which is toxic when inadvertently injected into the peritoneal cavity may produce direful results to the patient in the form of shock, perforation of the bowel and peritonitis. A solution which is unsterile will result in superficial abscess or peritonitis in the case of a perforation of the peritoneum. Lastly, the clinical evaluation on patients must be made and the results observed over at least a two year period in order to confirm the efficacy of the solution definitely.

Up to the present time these four criteria have been applied to three solutions, A, B and C, which have been used in our series. A and B are alcoholic solutions of tannic acid, and C is the salt of the fatty acids of oil of psyllium. Work on these three solutions has been done in identical experiments, and all have been definitely proved to produce fibrosis. There is apparently no difference in the speed of formation of the fibrous tissue nor in its density with any of these solutions. Experimental work on the injection of these solutions into the peritoneal cavities of animals reveals no evidence of inflammatory changes. However, in guinea-pigs, large doses of over 2.5 c.c. of any of these solutions will result in shock and death. This reaction is considerably less with solution C. All three solutions have been tested clinically, 75 patients having been treated with each solution. The results with all are equally good, and the recurrences are in no way dependent on which of these three solutions is used. Parenthetically, we might say that there is no special virtue or so-called "something" to a muchly advertised foreign solution when compared to the American made solutions studied by the authors. The danger of using solutions not experimentally and clinically proved must be emphasized repeatedly and a warning must be given to surgeons using this procedure against the many solutions placed on the market that have not been so proved.

The proved solutions are uniformly efficacious regardless of the age of the hernia, but generally speaking, all of them are less active as the patients are older. The speed of proliferation is slower in those of advanced age and more injections are necessary. Failure to consider this point will result in recurrences in aged patients.

A number of constitutional diseases slow down the rate of fibrous tissue proliferation and make more injections necessary. In severe cases this reduction in the rate of fibrous tissue formation may make the closure of the hernia not only difficult but often impossible. These include diabetes mellitus, syphilis, tuberculosis, nephritis and malignancy. While the experienced operator may attempt the cure of hernia in patients with any of these conditions, the prospects of failure and recurrence are definitely increased.

The Choice and Application of the Proper Truss. A prime cause of recurrence with this method is the use of a truss which fails to hold the hernia in reduction at

all times. This depends to a large extent on the type of truss used, and the factors upon which this selection is made, have been described. However, with the proper type of truss selected, certain conditions make the wearing and fitting of even such a truss difficult. In patients who are obese, the fitting of a truss is associated with considerable difficulty since the thick fat of the abdominal wall prevents the approximation of the muscle layers by reducing the available pressure of the truss. In such patients, the truss apparently holds the hernia closed, but actually leaves a small part of the sac contents within the sac, and predisposes to failure or recurrence. One of two procedures should be used before the injections are attempted. Either the patient should be placed on a reducing diet and an appreciable amount of weight lost, or a very large tight truss pad should be fitted and worn until a pressure-atrophy of the fat over the inguinal region results. Treatment may then be started as the truss will maintain reduction of the hernia.

In patients with enlarged abdomens there is usually a marked increase in the intra-abdominal pressure. This not only may be a predisposing factor to the formation of the hernia, but it also makes the application of a truss more difficult since ordinary trusses cannot hold the hernia in reduction under such circumstances. Special care in the selection of the proper truss must be given to these cases, and treatment prolonged if recurrences are to be avoided. Similarly, in any situation where the intra-abdominal pressure is increased, such as hypertrophied prostate and chronic constipation, extra care in regard to the truss and prolongation of the treatment must be maintained. These conditions make recurrences following the surgical treatment of hernia more likely, and likewise do so when the injection treatment is used. The fact that this is true must not be held against the injection therapy.

A minor but frequent complication is the excoriation of the skin under the truss pad during the first few days following the application of the truss. This can be avoided by the gradual application of the truss, that is, for a few hours each day until the truss is worn continuously. Such excoriations as do occur are treated with a pyrol and paracene mesh dressing. Many patients complain of pain over the back when certain types of trusses are worn. This can be obviated by the use of the proper truss.

The proper truss, improperly fitted, will fail to retain the hernia and will result in a failure in the treatment. It is wise for the surgeon undertaking this treatment to work with a competent truss fitter to correct this difficulty. Where none is available the operator must do the fitting himself. It is not the purpose of this article to discuss this phase of the subject.

Apprehensive and nervous patients will complain of the difficulties of wearing a truss, and if their cooperation is not assured, they are best not treated by this method. Similarly, some children, and especially young ones, will continually move the truss and allow the descent of the hernial contents, thus nullifying the treatment.

A question that has been frequently discussed as a contraindication to the injection treatment is that of atrophy of the testis following the treatment. Careful check-up on the patients in our series prior to the start of the injections reveals atrophy of the testes in 9.1 per cent of cases. It is felt most definitely that this atrophy is due to the use of an improperly fitting truss which causes pressure on the spermatic cord as it passes over the pubic bone. There has not been one case of atrophy of the testis following the injections in 225 cases in our series.

The Technique of Injection. In a consideration of any surgical procedure the first care must be toward the proper sterilization of the instruments used. Unsterilized instruments, or improperly sterilized instruments, will result in abscess formation at the site of the injection.

While critics of this method have repeatedly stressed this complication, ordinary care should prevent it. There has not been a single abscess in over 4500 injections in our series. Even in patients with diabetes mellitus, who are characteristically more susceptible to infection, this complication has not been seen.

The principal difficulties which may arise due to errors in technique are those which occur from placing the injections either too superficially or too deeply. If the solution is deposited in the fat, superficial to the external oblique muscle, hard, painful nodules appear in the injected area. These respond readily to heat and are absorbed in a few days. However, such injections will give no fibrous tissue to aid in the closure of the hernia and a sufficient number of properly placed injections must subsequently be made.

Injections that are placed too deeply are of two kinds: (1) those injected into the cord, and (2) those injected into the peritoneum. Injections which are placed too deeply into the cord result in increased pain and swelling of the cord within two to three hours after the time of injection. This may be associated with some edema and swelling of the penis, scrotum and epididymis, but such swelling usually disappears within twenty-four to forty-eight hours. Such swellings occasionally occur even when the cord is not entered, and are probably due to stasis from pressure of the newly formed fibrous tissue. Rarely too deep injections will result in hydrocele of the cord; 3 such instances occurred in our series. Drainage of the hydrocele and the injection of a suitable sclerosing solution will result in cure. Rarely, also, painful swelling of the epididymis has appeared which usually responds in a few days to heat and elevation with a suspensory, although in one case the pain persisted in a moderate degree for three months. This occurred in four patients. It must be stated that a certain amount of pain and transient swelling will occur with almost every case. The discomfort is not severe but must be expected.

A more alarming complication, but not more serious, is seen when injections are placed too deeply into the peritoneal cavity. A condition of severe shock ensues which is directly proportional to the amount of solution injected. Fortunately, the alcoholic preparations, Solutions A and B, give immediate pain when inadvertently injected into the peritoneal cavity and the injection may be stopped. The usual amount of solution injected is about 0.5 c.c.

Of particular danger in the use of Solution C is the property which makes it most adaptable for use, that is, its painlessness. When injected intraperitoneally, through an error in technique there is no immediate pain, and even though the usual 3 c.c. dose is injected slowly during the course of a two to three minute interval, the patient will complain of no pain, despite the fact that the fluid is already in the abdominal cavity. After about a five to ten minute latent period, however, the patient does complain of severe symptoms.

The episode, which is the same for all three solutions, is ushered in by gradually increasing abdominal cramps which may remain localized to the lower abdomen, or may spread to all parts of the abdominal cavity. The pain is cramp-like, quite constant, and is associated with pain in both testes, usually worse on the side of injection, pain in the penis, which is extraordinarily severe, and lancinating pains in both thighs, and toward the rectum. The pain becomes progressively worse throughout a period of about twenty to twenty-five minutes, during which time the patient complains of being chilly, and there may be a moderate or severe chill. The patient complains of pain in the extremities, both of the arms and legs, which is variously described as "a burning in the veins," "electricity in the arms and legs," etc. Following the chill which may last from five to ten minutes the patient

becomes diffused with a feeling of warmth and the pain gradually subsides during the course of the next half hour. The first pains to go are the abdominal cramps, with the penile, rectal and testicular pains leaving in that order. There is a residual soreness which remains over the site of injection. The patient is left with a feeling of weakness which gradually disappears in the course of the next three to four hours, and apparently there are no sequels from this mishap.

Examination during the course of the attack reveals the following: The patient lies on his back with the legs slightly drawn up, and while apparently in severe pain, moves only very little. Temperature is normal, respirations 35 to 50 and extremely shallow, pulse rate 100 to 120 and very weak in quality. The skin is pale and moist, and the face is covered with fine perspiration. The skin is cold and clammy to the touch. The face is pale and drawn, usually there are two bright red spots on the cheeks. The abdomen is soft, and no rigidity is present, but marked tenderness on deep pressure, especially over the lower abdomen, can be elicited. The penis and testes are superficially tender to the slightest touch.

The treatment consists in keeping the patient flat and applying hot blankets and hot water bags to the abdomen, genitalia, thighs and perineum. Morphine sulphate, grain ¼, may be given but the after effects of the drug cause more distress during the rest of the day than the aftermath of the injection.

A comparison of Solution C with Solutions A and B in this regard is difficult. Injection with these two latter drugs is immediately painful on entering the peritoneal cavity and injection of 3 c.c. of these solutions into the peritoneal cavity is not often made. If made, however, a similar picture ensues in a much more severe form, in fact the shock is so severe that life seems threatened. However, with ordinary care only a few drops of these solutions are injected, since pain cautions against continuance of the injection. The resulting clinical picture of pain and shock lasts only ten to twenty minutes. In general, then, while equivalent amounts of Solution C will give less general reaction than the same amount of Solution A or Solution B there is more likelihood of injecting a larger amount of Solution C with more painful results. This mishap has occurred seventeen times in our series, more frequently during our earlier experience with the method. No lasting harm has been seen and all of the patients were well within four to six hours after the injection. A good criteria of the severity of these and other complications is the fact that only twenty-one working days have been lost to this group of patients during the entire period of this study.

Another factor which should be considered under the technique of the injection is the question of perforation of a vein with the needle. Previous to the injection of the solution the plunger of the syringe is withdrawn to ascertain this fact. If blood returns through the needle, no injection is given that day. Hemophilia is a contraindication for this reason. Puncture of a vein has occurred once in every seventy-five injections in our series, but without any sequels.

In giving the preliminary novocain anesthesia, before injecting one of the alcoholic solutions to patients with hypertension, there may be complaints of dizziness or headache, especially if adrenalin has been used with the novocain. This causes only mild discomfort usually, but can be avoided by using a solution which requires no preliminary anesthetic.

Placement of the Injections. As stated the proper placement of the injections depends on a proper diagnosis of the type of hernia present. The importance of this point has been stressed. Starting injections at the internal ring in an indirect hernia, while technically more satisfactory, apparently does not influence either the percentage of recurrences or failures. The first seventy-five cases of indirect inguinal

hernia in our series were treated by starting the injections at the external ring. The factor of prime importance seems to be the constant reduction of the hernia by the proper truss.

Outline of Treatment. A prime cause of failures and recurrences by this method is insufficient treatment. Especially in older patients the slow rate of fibrous proliferation and the excessive resorption of the newly formed tissue make adequate and prolonged treatment imperative. A close adherence to the outline presented will minimize recurrences and failures from this cause. It is of utmost importance to realize that a sufficient number of injections must be given. Similarly, it is of importance to wear the truss for a sufficient length of time following the completion of the injections. Experimental work shows that the process of fibrosis continues for three to six months following the injection of the solution and support of the inguino-abdominal region by means of a truss must be provided during this period.

Certain commercial exploiters of particular solutions used in this treatment have made attempts to minimize the number of treatments necessary and to shorten the period of truss wearing. This has been done in an effort to make the method more desirable to both patients and physicians. In other words, the simpler the procedure, the wider its application. This attitude must be most heartily condemned. Experience with carefully followed patients shows just the opposite. This treatment can be successfully undertaken only with extreme patience and care. The fitting and care of trusses requires a number of days and frequent examination. The treatments themselves are exacting and require skill. The number of injections necessary are by no means small, and the period of follow-up requires constant watching of the individual case. The numerous possibilities for dangers and complications make continuous vigilance necessary. Time and an appreciation of the difficulties involved are necessary if recurrences and failures are to be avoided.

To summarize, the causes of recurrence and failure in the injection treatment of

TABLE IV

ANALYSIS OF RECURRENCES

Case	Group	Cause	Period of Time between Removal of Truss and Recurrence, Months	Present Status
1	Good risk	Improper truss.	1	Retreated and closed.
2	Good risk	Improper truss; incorrect diagnosis.	4	Retreated and closed.
3	Good risk	Improper truss.	3	Retreated and closed.
4	Good risk	Insufficient treatment.	5½	Retreated and closed.
5	Good risk	Insufficient treatment.	4½	Retreated and closed.
6	Poor risk	Constitutional disease (syphilis, under treatment); insufficient treatment.	7½	Retreated and closed.
7	Poor risk	Insufficient treatment.	4	Retreated and closed.
8	Poor risk	Insufficient treatment.	3½	Under treatment.
9	Poor risk	Constitutional disease (syphilis, under treatment); insufficient treatment.	6	Unable to close. Hernia one-third previous size.
10	Poor risk	Insufficient treatment; improper truss.	4	Under treatment.
11	Poor risk	Improper truss; incorrect diagnosis.	1½	Retreated and closed.
12	Poor risk	Improper truss; obesity.	2	Unable to close.
13	Poor risk	Improper truss; insufficient treatment.	8	Retreated and closed.
14	Poor risk	Enlarged abdomen; improper truss; insufficient treatment.	3	Under treatment.

hernia depend on neglect of the fundamental principles on which this treatment is based. Incorrect differential diagnosis of the types of hernia will result in improper fitting of the truss and the incorrect placement of the injections. Inefficient solutions will fail to give fibrosis. Proved solutions will act slowly in patients with constitutional disease. The improper type of truss will fail to maintain reduction of the hernia and will thus result in failure. Even the proper truss will be difficult to maintain in position in obese patients or in those with increased intra-abdominal pressure from any cause. The proper truss incorrectly fitted will result in failure. Apprehensive or nervous children or adults will move the truss, will be uncooperative and will make cure unlikely. Lastly, insufficient treatment and insufficient wearing of the truss will result in recurrence and failure even though all other factors are properly considered. An analysis of the causes of the 14 recurrences in this series is given in Table IV.

TABLE V
ANALYSIS OF COMPLICATIONS

Complication	Number	Percentage
Excoriation of skin under truss prior to injections.	17	7.5 of cases
Swelling of the cord.....	37	16.0 of cases
Transient swelling of penis, scrotum or inguino-abdominal region	203	90.2 of cases
Hydrocele of the cord...	3	1.3 of cases
Swelling of the epididymis.....	4	1.7 of cases
Fat nodules from superficial injection.....	3	0.06 of injections
Intraperitoneal injections	17	0.37 of injections
Needle in vein.....	60	1.3 of injections
Abscess.....	0	0
Peritonitis.....	0	0
Atrophy of the testis....	0	0
Impotence.....	0	0
Deaths.....	0	0

Of the many possible complications mentioned, only a limited number have occurred in the cases which we have treated; Table V summarizes these complications.

DISCUSSION

An analysis of the results in this series to date serves to illustrate two essential points: (1) that the method is of value and (2) that it involves extreme care on the part of the surgeon if good results are to be expected. A recurrence rate of 4.1 per cent in good operative risk patients we feel to be encouraging. The fact that a favorable prognosis can be offered to 80 per cent of the patients in the inoperable group is most satisfactory. Even in those cases which do recur, further treatment can be instituted following elimination of the cause of recurrence. Furthermore, such recurrences as did occur were much smaller in size than the original hernia and were more easily controlled by a truss. While a number of complications have occurred these have all been of a minor nature. There has been no mortality from this procedure.

On the other hand, strict observance of the many details of this procedure are necessary if recurrences and failures are to be avoided. The exacting nature of the treatment makes care of these patients a nuisance and a hardship for the busy practitioner. Even in the hands of competent men this method may be found unsatisfactory for these reasons. Recently in an editorial in the *American Journal of Surgery*, Coley stated that "first the results seemed very satisfactory but by the end of six months there were so many recurrences that the method was definitely abandoned."[12] Thus he summed up the experience with the injection method, of workers at the Ruptured and Crippled Hospital in New York. A statement of this type illustrates the difficulties that will be encountered.

In contrast to this is the dangerous, unethical and commercial attitude of the distributors of certain solutions used in this treatment. In their haste to exploit their own product many exaggerated claims are made both for their particular solution and for the method. Such tactics can lead

only to discredit of the entire treatment and must be condemned.

A sane middle course between these two extremes must be taken. The recent reports of careful investigators show experience with this treatment very much like our own. Numerous workers in this country, and Gray[3] in England have reported hundreds of carefully followed cases and all of them are making a sincere attempt to evaluate their results, and properly are refraining from premature condemnation or praise of this clinical experiment.

SUMMARY

1. The principles upon which the injection treatment of hernia is based are described.

2. The relationship of violation of these principles to recurrences and failures with this method is discussed.

3. The placement of injections and the necessary outline of treatment is emphasized as a means of reducing the recurrence rate.

4. The results of treatment in 225 cases of direct and indirect inguinal hernia are given; a recurrence rate of 4.1 per cent for good risk cases and 19.5 per cent for poor risk cases is reported.

5. A detailed analysis of the causes of recurrences, failures and complications is given in relationship to the fundamental principles of this treatment.

6. The complications which have occurred in this series are statistically evaluated and described.

7. The results herein reported warrant continuance of this work. The exacting nature of the technique and the numerous details involved in the care of the patient predispose to unsatisfactory results unless much time and care are spent by the surgeon. The unethical and commercial exploitations shown by distributors of certain solutions in an effort to minimize the complexities of this method must be condemned.

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EXTRADURAL HEMORRHAGE*

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THE subject of extradural hemorrhage is one of the most fascinating bypaths of head injuries. Here is a condition in which dramatic results may be obtained by careful observation, accurate diagnosis and skilful treatment. The peculiar syndrome of this complaint must have been manifest since the earliest times, when one man learned to club another, or even earlier, when falls from a height were possible for our arboreal ancestors. The general practitioner, and even the surgeon, is constantly on the lookout for an extradural hemorrhage and he is frequently harassed by the fear that he may overlook it. In some textbooks of surgery this subject does not appear to be discussed in enough detail and in others the description is actually misleading. The intricacies of the subject are interesting and their perusal instructive.

HISTORICAL NOTE

About a hundred years ago before anesthesia was discovered, one J. Hill, surgeon of Dumfries, Scotland, operated on 2 cases of extradural hemorrhage, one in the year 1750 and the other in the year 1751. Both patients were well in 1771, some twenty years later, though one of them is "liable to convulsive twitchings, but they do deprive him of his senses, and his memory at times fails him so far as to make him at a loss for words to express himself." Hill was evidently a man of no mean judgment and ability for practically two hundred years later our results are not strikingly better.

In 1816 Sir Charles Bell published a short monograph entitled "Surgical Observations" and in this book he discusses the mechanism by which an extradural hemorrhage is produced,

> It is extraordinary that anyone who has ever raised the skull cap in dissection, and felt the strength of the universal adhesions of the dura mater and the lower surface of the bone, could for an instant believe that arteria meningea media has the power of throwing out its blood to the effect of tearing up these adhesions from the entire half of the cranium! Strike the skull of the subject with a heavy mallet; on dissecting you find the dura mater to be shaken from the skull at the point struck. Repeat the experiment on another subject, and inject the head minutely with size injection, and you will find a clot of the injection lying betwixt the skull and dura mater at the part struck, and having an exact resemblance to the coagulum found after violent blows on the head. I imagine this is conclusive.

In 1842 Cock of Guy's Hospital, gave a very clear description of a typical case of middle meningeal hemorrhage. The patient was admitted at 7:00 P.M., September 2, 1841; at midnight collapse had passed off and he seemed rational; at 4:00 A.M. he got out of bed to relieve his bowels, and at 8:00 A.M. he was stuporous; by noon he seemed in extremis, but Cock operated and removed the clot and he ultimately recovered.

Johathan Hutchinson in 1867 drew attention to the dilated pupil often to be found on the homolateral side. The condition has come to be known as a Hutchinson pupil. He pointed out that the side of the clot could be determined by (1) hemiplegia, if present, (2) dilated pupil, usually on the same side, and (3) puffy swelling of the scalp found directly over the fracture. He emphasized the impor-

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tance of the previous history. Hutchinson was very pessimistic about trephining in cases of middle meningeal hemorrhage. Under the heading of effusion of blood between the bone and dura he says, "These are especially important, because generally supposed to be capable of treatment. Yet it is a remarkable fact that the modern annals of surgery do not, as far as I am aware, contain any cases in which life has been saved by trephining for this state of things." He laid stress on the importance of a history of lucid interval.

About the same time Callender stated that he agreed with Hewett about the uselessness of trephining in these cases and he added that for the last six years the surgeons at St. Bartholomew's Hospital had discontinued the practice.

In 1873 Gross in America drew attention to middle meningeal hemorrhage and its treatment. He was at that time engaged in military practice and his first case presented itself after the battle of Shiloh, April 6, 1862. He reported 8 cases of which 7 were direct head wounds by rifle balls. He was strongly of the opinion that an extradural clot should be immediately evacuated. In addition he stated that not only extradural but intradural clots should be evacuated.

In 1885 Jacobson wrote his classical description of middle meningeal hemorrhage and, in my opinion, this must be regarded as the most complete and thorough work on the subject. In this article the subject was taken up in all its aspects and 70 cases were reported and discussed; 27 cases were from Guy's Hospital and 43 were reported from the English literature. At the time the article was written Jacobson must have had the opportunity of studying some 4 or 5 cases. This article will always remain the classical description for there is very little that can be added to it. Some new points have been brought up in the last fifty years and certain old points need re-emphasizing.

In 1886 Kronlein wrote a monograph based on the study of 280 cases, but for directness and lucidity it does not compare with the work of Jacobson.

SOME ANATOMICAL CONSIDERATIONS

The middle meningeal artery enters the skull through the foramen spinosum and is accompanied by the nervus spinosus, i.e., the recurrent branch of the mandibular nerve and an accompanying vein. The accompanying vein is said to communicate with the pterygoid plexus (MacEwen). The vein must be very small and inconspicuous because in operations for trigeminal neuralgia in which the middle meningeal artery is tied intracranially at the foramen spinosum, the vein is rarely seen and very rarely bleeds. The artery is subject to wide variation within the skull. Plummer in 1896 made some researches into the surgical anatomy of the artery, during which he studied 50 skulls. He stated that in 95 per cent there was a main trunk but in one the artery divided into anterior and posterior branches at the foramen and in another the artery came through two foramina, one in front of the other. Occasionally the ophthalmic artery is replaced by a branch from the middle meningeal. Usually the artery runs for a short distance, an inch or so, and then divides into an anterior and a posterior terminal branch. Before it branches, however, the artery gives off several small branches, a superficial petrosal which enters the hiatus canalis facialis (hiatus Fallopii), branches to the semilunar (Gasserian) ganglion, a superior tympanic branch, and an orbital branch which may arise from the anterior branch of the main artery and which enters the orbit through the superior orbital fissure (sphenoidal fissure) and anastomoses with the lacrimal artery. The point of bifurcation of the artery is said to be about one-half inch above the middle of the zygomatic arch; but if the main trunk is short, the bifurcation may take place at or below the arch. The *anterior branch* is the larger of the two and runs anterolaterally along the greater wing of the sphenoid to the sphenoidal

angle of the parietal bone, where it is embedded in a deep groove or even in a body tunnel. It ascends behind the anterior border of the parietal bone toward the vertex, that is, parallel to and slightly in front of the motor cortex; branches pass forward and backward from it. The *posterior branch* passes posteriorly from the greater wing of the sphenoid to the squamous portion of the temporal bone where it travels almost horizontally backwards, sending branches to the posterior fossa and upwards to the vertex. As has been said before, the artery is subject to as wide variability as the skull in this region. The artery may pass through a bony tunnel, a deep or a shallow groove. In approaching the artery from the lateral aspect along the floor of the middle fossa various abnormalities of the bone may be found, notably a large "hump" situated anterolaterally to the foramen spinosum and which may altogether obscure it. In 2 recent cases in which the ganglion was being approached for the relief of trigeminal neuralgia, the artery was absent; there was a small foramen spinosum.

The grooves on the inner surface of the skull in the region under discussion are usually believed to be due to the pulsating middle meningeal artery, but as a matter of fact they are produced by the veins which accompany the artery. This was pointed out by Wood Jones, but as he says the earliest English anatomists were aware of this. Thus Helkiah Crooke in 1651 referring to the bones of the sinciput said, "These bones have on the inside superficial cavities or small bosomes to which the Dura Meninx grows, as also inscriptions for the vessels which run from the internal jugulars through the thick membrane of the brain. They also have small perforations, especially outward at the sides of the Sagittal suture which perforates the bone from one side to another, and transmit small veins for nutrition." Thomas Gibson in 1688 talking of the bones of the sinciput wrote, "On the outside they are smooth, but on the inside uneven, for they have a great many furrows running along their inner superficies for the passage of the veins of the dura mater." However, James Keill in 1723 made the first mention of the grooves being caused by pulsating arteries and from that time the idea has been held by most surgeons. He writes, referring to the parietal bones, "They are smooth and equal on their outside, but on their inside they have several furrows, made by the pulse of the arteries of the dura mater."

There is, to say the least of it, a striking similarity between the sentence just quoted by James Keill and that written forty years before by Thomas Gibson; only the interpretation is different. In 1911 Wood Jones again drew attention to these grooves on the inner surface of the parietal bone and showed that they were formed by the sinuses running with the artery. This he stated to be the case for the following reasons: (1) compare the bore of the foramen spinosum with that of the so-called arterial markings; the markings are often two or three times the size of the bore of the foramen spinosum; (2) these markings do not converge to the foramen spinosum; (3) they do not become finer and finer as they pass away from the foramen; on the contrary (4) cross sections through the grooves demonstrate clearly that the grooves contain the sinuses and that the artery does not lie in contact with the bone. This view has been accepted by anatomists but does not appear to have been fully realized by surgeons, though it is of some practical surgical importance.

It is well to remember also that the skull is thinnest in the temporal fossa and especially over the squamatemporalis and that the anteroinferior angle of the parietal bone is very brittle, there being practically no diploë in this region. In addition it is said by some (Tillaux) and inferred by others (Bell) that the dura mater is particularly easy to detach from the bone in this region. This may be true of adults but it is not true of children and very

old people. The underlying brain in the region of the middle meningeal artery is also of importance, the anterior branch

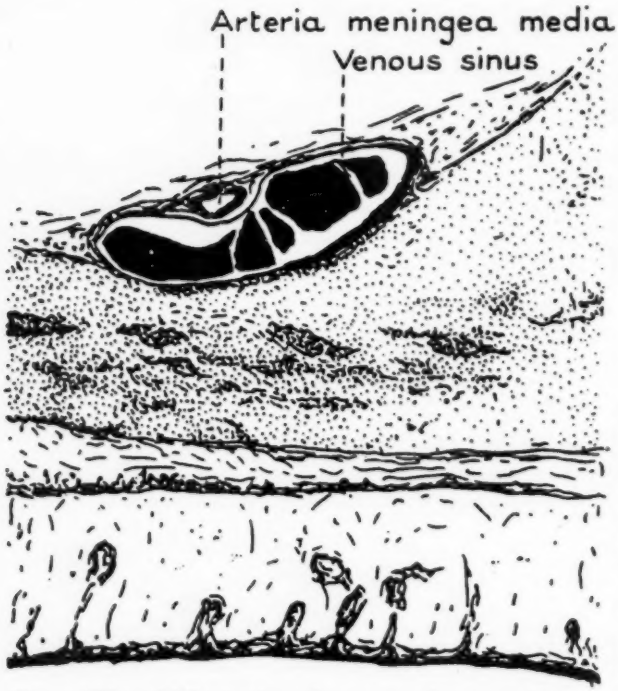

Fig. 1. The middle meningeal artery and venous sinuses showing the protected position of the artery and the exposed position of the sinus (*after Wood Jones*).

practically overlying the motor area and on the left the speech center.

There are also certain peculiarities of the child's skull concerned in fractures (Fraser): (1) The bones are more elastic and tend to bend rather than break. (2) The large amount of cartilagenous or fibrous tissue tends to limit transmission of the injury. (3) The diploë is largely absent and therefore fractures of the inner table alone are rare. (4) Comparative absence of air sinuses in the child makes a frontal fracture more important. (5) Adherent dura tends to limit extradural effusions.

The meningeal veins begin in two capillary plexuses, a deep and a superficial. The deep plexus is a wide network in the inner layer of the dura mater; its efferents terminate in the superficial plexus. The superficial plexus lies in the outer layer of the dura mater. It consists of numerous vessels of uniform caliber which frequently anastomose and terminate in two sets of efferents; of these, one set ends in the cranial blood sinuses and the other accompanies the meningeal arteries. The efferent meningeal veins are peculiar inasmuch as they do not alter much in size as they approach their terminations. The middle meningeal veins receive communications from the sphenoparietal sinus. They lie external to the arteries in the grooves in the inner wall of the cranium and are very liable to be torn when the bones are fractured (Wood Jones).

PATHOLOGICAL CONDITIONS

LeCount and Apfelbach studied 504 cases of trauma in which there was a fracture of the skull demonstrable at autopsy. Many other studies of this kind have been made but this one has been selected because of the writer's close association with one of the authors. There was evidence of extradural hemorrhage in 199 of 504 in this series or 39.48 per cent; of these, 104 were large enough to have caused appreciable compression and 95 were small. Of the 104, 73 or 70.19 per cent were associated with fracture of the middle fossa, 15 or 14.42 per cent with fracture of the posterior fossa, 10 or 9.61 per cent with fracture of the vault and 3 or 2.88 per cent with fracture of the anterior fossa. Postmortem examination in these cases showed that the extradural hemorrhage was on the left side 54 times and on the right 49 times, the hemorrhage covered the temporal and parietal lobes in 49, the temporal, parietal and occipital lobes in 34, the occipital lobe in 14, the frontal and parietal lobes in 6, and the temporal and parietal lobes on both sides in 1 case. The bleeding was attributed to the anterior branch of the middle meningeal artery in 49 cases, to the posterior branch in 44, in 3 cases to a laceration of the superior longitudinal sinus and in 8 cases in which the fracture was limited to the posterior fossa the bleeding was considered to have arisen from tearing of the lateral sinus. The condition of the underlying brain was also examined particularly for bruises; of the 67 cases

of fracture of the middle fossa that were examined, in 9 cases no bruising was seen, in 33 cases there were superficial bruises, and in 25 cases the bruising was marked; there were bruises in all 15 cases of posterior fossa fracture.

Moody studied 908 cases of head injury in which skull fracture was verified either by autopsy or by operation in 547 cases. In this series of 908 cases, there were 100 instances of middle meningeal hemorrhage of such size that compression of the brain by blood was the chief cause of death. This was confirmed in all the cases by operation, autopsy or both. Diagnosis was not made clinically in 63 cases of the mentioned middle meningeal hemorrhages; in 37 the condition was recognized and operation performed; of these 37 patients, 26 died, 21 being operated on within twelve hours. This study by Moody verifies rather strikingly Frazier's contention that, "Almost 50 per cent of massive middle meningeal hemorrhages are overlooked." Moody also gives some specific information as to the weight of the clot in the 67 cases that came to autopsy; he found that the maximum weight of the clot was 246 grams, the minimum 40 grams, and the average 110.5 grams.

F. Wood Jones studied at postmortem 3 cases in which death had been caused by massive middle meningeal hemorrhage. The tissues were studied by injection of dyes, gross examination and serial sections. In all 3 the venous sinuses were injured but not the artery. This point was hinted at in the discussion of the anatomical considerations, where it was noted that the accompanying venous sinuses lie in the grooves of the parietal bone external to the artery and the more friable veins are thus more exposed to injury.

In some cases extradural hemorrhage has occurred without it being possible to demonstrate a fracture of the cranial bones. In these cases I suppose that, as in Sir Charles Bell's experiment, the dura was shaken away from the skull by the blow; and should the artery and accompanying sinuses be deeply imbedded in a groove or even in a tunnel, a tear would take place at the point of emergence of the vessels, i.e., where the dura that was moved by the blow joined the dura that was fixed in the groove or tunnel. Other writers believe that the sphenoparietal sinus (Trolard) is frequently injured. Here again there is a communication between the meningeal veins in the "movable" dura and the fixed sphenoparietal sinus (see anatomy). These factors have a distinct bearing on the diagnosis and treatment of the condition under discussion.

SYMPTOMS AND SIGNS

The classical syndrome of extradural hemorrhage is characterised by violence to the head with unconsciousness followed by a return to consciousness for a few hours with a subsequent return to unconsciousness. The diagnosis is established mainly by the lucid interval followed by a return to unconsciousness. Very little emphasis is placed on the absence of the lucid interval and on the presence of hemiplegia.

Case 1. E. W., a forty year old white man was admitted to the examining room of the Cook County Hospital, January 21, 1934 at 11:20 A.M. in coma with the odor of alcohol on his breath. He was said to have "fallen" in a police cell where he was "found" with a bleeding nose. He was admitted about one-half hour after the injury. The pulse rate was 76, temperature 98 degrees F. by rectum, and respirations 16. On physical examination nothing abnormal was found except that the nose was filled with coagulated blood. There was no apparent bruise or laceration of the scalp. In the examining room a diagnosis of cerebral hemorrhage was made, but in the ward it was felt that alcoholic intoxication or middle meningeal hemorrhage was more likely. At 5:30 P.M. the patient was awake but drowsy and complained of pain in the right side of the head. He talked with great difficulty and was unable to account for his accident. On re-examining the patient it was noticed that he had a left hemiplegia with slight movement in the left leg, sensation seemed to be diminished on the left, the reflexes were

exaggerated, there was some ankle clonus but no Babinski; the pupils were equal and reacted to light. A diagnosis of middle meningeal hemorrhage was made and I was asked to see him.

The patient was semistuporous. Spinal puncture revealed bloody fluid with a pressure of 20 cm. of spinal fluid. There was paralysis of the left arm and the left side of the face, the knee and biceps jerks were exaggerated on the left. The left plantar response was questionable, the right being normal. There was evidence of left hemianesthesia. It was suggested that a burr hole be made under local anesthesia.

At operation the following day a large right sided extradural clot was discovered, its lowest border being at the upper border of the squama temporalis. It was judged to be 10 cm. in anteroposterior direction, by 10 cm. in a coronal direction and about 3 to 4 cm. in thickness. No bleeding vessels were seen or ligated and the wound was packed with two iodoform gauze drains.

The next day the patient moved his left hand freely and was quite free from stupor; his condition was very good. On January 24, one pack was removed and followed by a gush of dark blood. On January 25, the second pack was removed and was again followed by a gush of dark blood and some fresh blood. The meninges pulsated freely, but the patient seemed rather drowsy and did not move his left hand as well as previously. A gutta percha drain was inserted down to the dura.

On January 26, the patient was comatose and hemiplegic so he was at once removed to the operating room. There the old wound was enlarged upwards exposing an anteroposterior linear fracture about 8 cm. above the zygoma with a large amount of subjacent extradural clot. This was removed and the wound repacked; no specific bleeding points could be found. The patient recovered consciousness on the table, and from then onwards made an uneventful recovery. He has been seen and heard of at frequent intervals to the present time.

It seems almost inconceivable that if all cases, or even the majority of cases, passed through this obvious and characteristic symptom complex, such statistics as those collected by Apfelbach and LeCount, and by Moody, could be possible. I believe one is justified in agreeing with Frazier that, "Almost fifty per cent of massive middle meningeal hemorrhages are overlooked." If this is the case, what can be done about it? Probably all or most of the cases presenting the classical syndrome are recognized, but there must be a large number of cases in which the classical syndrome does not appear, being masked by injuries to the underlying brain. Yet the diagnostic difficulties are not always insurmountable. When the signs of the developing extradural clot are obscured by injuries to the brain, the gradual onset of hemiplegia with deepening coma are reliable guides. Occasionally also the Hutchinson pupil may be of service.

In the following case it was possible to make a diagnosis of extradural hemorrhage in the absence of a lucid interval.

Case II. J. R., a fifty-three year old white man was admitted to the dressing room of the Cook County Hospital March 29, 1933, unconscious, incontinent and supposed to have consumed a large quantity of alcohol. He was brought in by the police twenty-three hours after the injury, and was said to have fallen downstairs while under the influence of liquor.

Physical examination revealed a well developed, well nourished white male, unconscious and incontinent. The temperature was 101.6 degrees F. by rectum, pulse rate 72, and respirations 20; blood pressure, systolic 150, diastolic 70. There was a depressed area palpable through the scalp in the right parietal region. There was clotted blood in the right ear. Both pupils were fixed and the right was larger than the left. The lungs, heart and the abdomen were normal. There were no obvious injuries to the extremities. The patellar reflexes were present. The patient was sent to the ward with a diagnosis of skull fracture and severe brain injury associated with alcoholism.

During the following day the patient's general condition remained much the same except that his temperature rose to 103 degrees F. by rectum, his pulse rate being relatively slow, about 84, and the respirations 24. On March 31 he was still in deep coma but his temperature had risen to 105 degrees F., the pulse rate was about 110, and the respirations 30 or more. At this time moist râles were

heard at the bases of both lungs. The reflexes were equal on both sides. He moved all his extremities but a bilateral Babinski was noticed. It was feared that a broncho-pneumonia was developing. Spinal puncture had been attempted on two occasions and had been unsuccessful.

Later that day I was asked to see him and the following note was made: "History of severe head injury fifty-two hours ago. This morning forty-eight hours after the injury the senior interne noticed he did not move his left side. He has not recovered consciousness since the accident. The left side is flaccid and he does not move it on stimulation. The left eye closes and seems fairly normal; the pupil reacts slightly to light. The right pupil is large and fixed and there is a divergent squint. The deep reflexes are increased on the left and there is a bilateral Babinski. Lumbar puncture between the third and fourth lumbar vertebra, reveals bloody spinal fluid (bloody III), pressure fairly normal, amount withdrawn 15 c.c. The intracranial pressure is not markedly increased and yet a hemiplegia has developed gradually. The patient's general condition is poor, he coughs and there is evidence of a beginning bronchopneumonia. The hemiplegia may be due to edema or to extradural hemorrhage. Recommend burr hole over the right motor area under local anesthesia."

During the forty-eight hours or so since his admission little had been done except observe him and to give about 2500 c.c. of fluid subcutaneously consisting of 1500 c.c. of normal saline and 1000 c.c. 5 per cent glucose.

At operation a large extradural clot about 5 cm. deep and about 12 cm. in diameter was exposed in the right temporal region. There was slight venous oozing after the removal of the clot, but the wound was closed without drainage.

On April 1 the patient was still comatose and he was receiving 400 c.c. of 25 per cent glucose twice a day. On April 2 he was seen to move the extremities on the left side. On April 3 considerable improvement was noted, the patient answered questions in a mumbling manner, and he moved his left side. His temperature was down to 101 degrees by rectum. On April 4, his temperature rose rapidly to 105.8 degrees by rectum with hurried and shallow respirations and a pulse rate of 124 to 136. The decompression was soft and pulsating. During the next few days his chest condition became gradually worse and on April 9 he died of what appeared clinically to be a bronchopneumonia complicated by a head injury. He continued to move the left side of the body and he responded and was cooperative, although by no means fully conscious, until he died.

The diagnosis in this case, and in the others which did not have a lucid interval, was established by close observation of the patient. First it was noticed that he did not move his left side as much as his right and later the presence of a hemiplegia was established by the absence of movement on supra-orbital pressure.

Case III. R. S., a twenty-two year old white male, fell off a motorcycle and was immediately taken to a doctor's office. When he entered the office he was quite rational but in the course of the next ten minutes he had a convulsive seizure and was immediately admitted Cook County Hospital, May 19, 1935 at 5:30 P.M.

On examination the temperature was 98 degrees F., the pulse rate was 88 and the respirations 24. There was a large contusion over the right parietal region. Some blood was oozing from the mouth and from the nose. He was said to have moved all his extremities. A diagnosis of probable skull fracture was made. The patient was unconscious and a spinal puncture revealed bloody fluid under increased pressure.

The next morning a further examination was made and an interview with the patient's sister brought out the fact that he had never previously had a convulsive seizure. At this time it was noted that he did not move the right side of the body as vigorously as the left. A large bruise the size of an egg was present behind and above the left ear. The patient was found to have difficulty in respiration although there were no pathological signs in the chest. The pupils were equal and reacted to light. The deep reflexes could not be obtained in the right upper extremity but the deep reflexes in the other extremities were equal and normal. There was slight engorgement of the retinal veins and blurring of the left optic disc. A diagnosis was made of left middle meningeal hemorrhage with basal skull fracture. I saw him a few minutes later and made the follow-

ing note: "Accident seventeen hours ago with deepening coma and right hemiparesis. Moved all four extremities on admission. Right facial weakness +3.4, right arm weakness +3, right leg weakness +3, the right foot lies everted. There are no movements of right side on supra-orbital pressure. Pupils left greater than right, the right reacts slightly to light but the left does not. Bilateral Babinski. Multiple bruises over the head; bloody spinal fluid. Fundi, right, normal; left, shows blurring of the nasal margin of the disc.

"*Diagnosis.* Severe brain injury and skull fracture with left extradural hemorrhage."

At operation a burr hole was made 1½ inches above the middle of the left zygomatic arch exposing the anterior inferior edge of an extradural clot. Another burr hole was placed just above and behind the ear, which necessitated a skin incision through the bruised area. The bone was fractured and the posterior branch of the middle meningeal artery was torn. Two arterial bleeders were controlled with silver clips but there was extensive venous bleeding. The clot was shelled out and the wound closed with two rubber tissue drains. The patient's condition was very poor. The prognosis was poor and the mortality quoted at 75 per cent. He died a few hours later.

Following is an abstract of the findings at autopsy:

1. Skull fracture with laceration of the posterior branch of the middle meningeal vessel on the left side, surgically sutured with silver clips.
2. Extradural hemorrhage over the frontotemporoparietal lobes of the left cerebral hemisphere, subdural hemorrhage over both cerebral hemispheres.
3. Cortical and subcortical laceration of the frontotemporoparietal lobes of both cerebral hemispheres.
4. Hyperemia and edema of the brain and lungs.
5. Cloudy swelling of the myocardium, liver and kidneys.
6. Septic softening of the spleen.
7. Stasis catarrh of the esophagus, stomach and duodenum.

Occasionally convulsive seizures, often of the Jacksonian type, may usher in the hemiplegia and coma as in the following case.

Case IV. S. T., a seventeen year old colored male was admitted Cook County Hospital October 27, 1935 at 12:30 P.M., about an hour and a half after receiving an injury to the head which was followed by two convulsions.

The patient was struck on the back of the head with a broom handle. He immediately sank to the floor holding his head and complaining of severe pain in the left frontoparietal region. He was fully conscious. About forty minutes after the injury he had a convulsive seizure during which he fell off a couch and injured his eye. About five minutes before entering the hospital he had a second convulsion. He was said to have become rigid first in the legs and then in the arms; later in the seizure he shook all over and gnashed his teeth. His mother stated that he had never had a convulsion before. About half an hour after admission the patient recovered consciousness and was quite clear and rational.

Examination showed a restless colored male who complained of tenderness in the left side of the head; temperature 98.4 degrees F. by rectum, pulse rate 100, respirations 24, blood pressure, systolic 112 and diastolic 78. There was tenderness in the left parietal region but no laceration, tumor or crepitus. There were abrasions on the left side of the face; the left upper lid was discolored and there was a subconjunctival hemorrhage without any posterior limit at the outer side of the left eye. The pupils were equal and reacted to light. There was an abrasion of the upper lip and the tongue had been bitten. Examination of the chest, abdomen and extremities did not reveal any abnormalities. An examination of the central nervous system was essentially negative. A diagnosis of concussion was made, and a note to rule out extradural hemorrhage was appended.

His condition remained unchanged all day.

The following day the tongue deviated to the right, and the other cranial nerves were normal. An examination of the nervous system was negative except for a marked motor aphasia.

October 29 another convulsion occurred. A spinal puncture was done and clear fluid was obtained under slightly increased pressure; the Pandy was negative and there were 4 cells per cm.

X-ray picture of the skull was negative but one repeated on October 31 demonstrated a linear fracture through the left parietal bone.

On November 1 the patient had several right sided convulsions in which there was

twitching of the right side of the face; the eyes deviated sharply to the right as did the tongue. Another spinal puncture was done and the fluid was still clear and under normal pressure. The following note was made: "Injury five days ago, became worse today with stupor, now almost comatose. Persistent right facial spasm with right facial weakness at rest. Increased deep reflexes on the right with fairly good power in the right leg though the right arm is paralyzed; no Babinski. The abdominal reflexes are diminished on the right but the cremasteric reflexes are equal. No response to pain in the right arm. The eyes deviate to the right, the pupils are equal and react sluggishly to light. Spinal fluid clear. *X*-ray film reveals linear fracture in the left parietal region.

"*Diagnosis.* Cortical irritation left motor area; rule out extradural hemorrhage. Advise burr hole with left subtemporal decompression."

At operation, an extensive extradural clot was found. One large meningeal vein was seen to be bleeding steadily; this was tied off as were several other small bleeding points. There was still some oozing from above under the bone and two iodoform gauze drains were inserted. The clot measured approximately 12 cm. by 10 cm. by 3 cm.

The next morning the patient was rational and moved his right arm freely. There was some blood on the dressing but it was decided to leave well enough alone. There was still an aphasia. That night, at 10:00 P.M., he began to have right sided convulsions again and in a very short time he was in coma. The wound was reopened in the operating room and some more bone rongeured away superiorly in the region in which the packs had previously been inserted. A small clot less than 1 cm. in thickness was found. There was some dural oozing in the superior portion of the wound which was probably from the same source as that mentioned at the previous operation. A large piece of muscle was stitched down on the dura in this region and the wound closed with one rubber tissue drain.

The patient then made an uneventful recovery and a month after operation, was back at school. He was seen in the clinic in October, 1936, and was in perfect health.

In some cases the arterial bleeding may be so severe and the bleeding point may be deep and so inaccessible that it is necessary to resort to tying the external carotid artery. This was done in the following case.

Case V. E. B., a forty year old white man was brought to the Cook County Hospital December 26, 1934 at 4:00 P.M. by the police with a history of having fallen downstairs and sustained a head injury fourteen hours previously. Following this the patient failed to respond although he had been very restless. The family had not seen him move his left side since the injury.

On admission the temperature was 99.2 degrees F. and the pulse rate 72. The blood pressure was systolic 132 and diastolic 80. There were two bruises on the right side of the face; the pupils were equal and reacted to light; there was a small amount of clotted blood in the right ear; there was some asymmetry of the face. Slight rigidity of the neck was noticed. The heart, lungs and abdomen were apparently normal. It was noticed that the patient did not move his left arm although the left leg was occasionally moved slightly. Urine examination was negative. A diagnosis was made of cerebral injury with localizing signs.

I saw the patient the next morning and from the story formed the impression that the hemiplegia came on immediately or very soon after the injury. A depression of the skull could not be felt. Emergency *x*-ray plates were reported as revealing no apparent fracture. On account of the history and the fact that the patient did not appear to be becoming more stuporous, a diagnosis was made of localized cerebral injury.

The next morning (December 28) at operation under local anesthesia an extradural clot, 8 cm. by 8 cm. by 2 cm., was discovered in the right temporal region. Overlying the clot the bone showed many linear radiating fractures. Two branches of the middle meningeal artery were ligated and the wound was packed with two iodoform gauze drains.

The following day the patient was conscious at intervals but at other times was irrational; the temperature rose to 104 degrees F. by rectum and the pulse rate to 120. He did not move his left side. On December 30, the patient improved. The next day he still could not move the left side although he was perfectly conscious. By January 4, he moved the left side a little and the drains were being shortened; by January 7, the drains were removed and the movements on the left were

much more active. The wound healed over but on January 29 it was bulging and fluctuating without pulsation; on incising the skin, dark blood gushed out and the wound was packed. On January 30 he was taken to the operating room because of continuous oozing from the wound, and when the packs were removed there was very free bleeding from the dura, but the bleeding point could not be found. It appeared to be in the depths of the wound near the foramen spinosum. The wound was packed again and he was given 500 c.c. of blood and the next morning the external carotid artery on the right side was tied above the lingual artery. From this point onward he made a steady recovery. He has been seen frequently and although he walks with a scarcely perceptible limp he feels perfectly well. The grip in the left hand is only slightly weaker than that in the right.

Case VI. W. T., was admitted to the Cook County Psychopathic Hospital June 10, 1936. Two days ago he was arrested in a drunken stupor. While he was in the cell he became disturbed and climbed walls and pounded on the doors. After he quieted down he was brought here, from the police station.

This information was given by the patient's wife who was extremely upset to find the patient in this hospital as she had never known him to have any mental disturbance. She added, however, that the patient was in the habit of going on periodic drinking bouts. The patient was admitted to the hospital on the order of a county physician as an alcoholic psychosis.

On examination, the patient showed many bruises about the head and body and he was bleeding from the left nostril; he was semiconscious and muttered unintelligibly. There was an ecchymosis of the left eyelid and an abrasion of the left side of the forehead and another abrasion in the left temporoparietal region. The temperature was 98.6 degrees F. by rectum, pulse rate 70, and the respirations 24. The right pupil was larger than the left, the left being somewhat irregular; both pupils reacted sluggishly to light. Dried blood was present at the nostrils; there was not any bleeding from the ears. The neck was not stiff. Examination of the chest and abdomen was negative. The right arm and leg were considered to be somewhat more spastic than the left and the deep reflexes were exaggerated on the right. The plantar reflexes were flexor in type. There were several bruises on the legs. The urine and blood were negative. A diagnosis was made of cerebral injury.

Two days later a spinal puncture was done and bloody spinal fluid was obtained. The patient's condition was not altered. The pulse rate had varied between 56 and 86, and the temperature had been about 99.4 degrees by rectum. A neurological examination revealed a right hemiplegia without Babinski, Oppenheim or Hoffman reflexes. The weakness did not involve the face. The deep reflexes were very active throughout but not more so on the right than on the left. The pupils were equal but the right pupil reacted more sluggishly to light. The pulse rate was 78. A diagnosis of subdural hematoma was suggested and instructions left to watch the patient carefully.

On June 16 I saw the patient and found his condition much the same as mentioned already; the pulse rate was 62 and the temperature 98.6 degrees by rectum. A neurological examination was made of which this is a transcription:

"The patient is disoriented as to time and space and may be aphasic. Profound weakness of the right arm +3.4, and marked weakness of the right leg +2.3, no weakness of the face, some movement of the fingers. The plantar reflexes are flexor, Hoffman's sign is absent, the abdominal reflexes are absent, slight cremasteric reflexes. Anesthesia can not be demonstrated. Pupils react sluggishly to light, the left less briskly than the right. The right pupil is larger than the left. The fundi are negative."

Because there was no weakness of the face, and because the patient's condition remained stationary, I felt that this was probably not an extradural hemorrhage. However, it was decided to put in a burr hole over the arm area on the left side of the head.

A linear fracture of the parietal bone on the left side was brought into view by the burr hole incision. A large extradural clot 10 by 10 by 4 cm. was found extending upward toward the leg area but not downward. When the clots were removed from the dura there was brisk arterial bleeding which could not be controlled by sutures as it came from branches down toward the floor of the middle fossa, which could not have been reached except by a wide craniectomy. The wound was packed and the left external carotid artery was tied. The patient made an uneventful recovery.

The peculiar distribution of the paralysis, the absence of pathological reflexes,

and the relatively non-progressive character of the lesion were confusing. A burr hole situated in the position commonly described in textbooks of surgery and of surgical anatomy would not have disclosed the clot; it would have been too low.

Case VII. E. W., a young white male, was admitted to the Cook County Hospital, June 24, 1936 at 7:00 A.M. in coma. He was picked up by the police one hour before and was said to have fallen off a porch. There was some swelling and discoloration of the left eye, and an alcoholic odor to the breath. The temperature was 99.6 degrees F. by rectum, the pulse rate was 92, the respiration 18, and the blood pressure 120 systolic and 78 diastolic. There was some blood in the left nostril. The chest and abdomen were negative. The reflexes were considered to be normal, but on supra-orbital pressure he failed to move the left side of his body as actively as his right. Spinal fluid pressure was increased and the fluid was bloody. The urine was negative. A diagnosis made of contusion of the brain and alcoholism. The next day his condition seemed to be worse and I was asked to see him. The following is a transcription of my note: "Pupils react sluggishly to light and the right is larger than the left. The abdominal reflexes are absent. The biceps and triceps jerks are not obtained. The knee jerks on the right are greater than those on the left. The left arm is completely paralyzed, the left leg is weak and there is slight weakness of the left side of the face. He responds to supra-orbital pressure. There is a bilateral Babinski sign. The pulse rate is slow, though it has been faster, and the temperature is 102.4 degrees F. by rectum. The signs have been progressive. The diagnosis is extradural hemorrhage on the right side of the brain, though brain contusion by contrecoup is possible. An immediate burr hole is advised."

A burr hole was placed in the right temporal region under local anesthesia supplemented with ether. No extradural hemorrhage was found. On incising the freely pulsating dura, 20 or 30 c.c. of slightly blood stained cerebrospinal fluid escaped. The temperature, pulse rate and respirations gradually rose and he died twenty-seven hours later with a running pulse and a temperature of over 106 degrees F.

The findings at postmortem examination are almost self-explanatory. "There is a hematoma in the deep scalp tissues over the temporofrontal area on the left side and temporal area on the right side. There was a 1.3 cm. surgical trephine opening in the temporal bone on the right side. There was a linear fracture of the skull which began in the parietal bone 1 cm. above its junction with the temporal bone and 1 cm. behind its midportion, on the left side. This fracture extended downward, lateralward and forward through the middle of the supraorbital plate on the left side to the cribiform plate of the ethmoid bone. There was clotted blood in the ethmoid sinus. There was a subdural hemorrhage over the frontotemporal lobes of the left cerebral hemisphere and temporal lobe of the right cerebral hemisphere. There was a subleptomeningeal hemorrhage over the temporoparietal lobes of the right cerebral hemisphere and cortical and subcortical hemorrhages in the inferior convolution of the parietal lobe and superior and middle convolutions of the temporal lobe of the right cerebral hemisphere. The cerebral convolutions were flattened. On multiple surfaces made by cutting the remainder of the brain, the cortex and basal nuclei were salmon colored, the white matter was pale and soft.

"*Summary*. Skull fracture as above; hyperemia and edema of the brain and lungs; cloudy swelling of the myocardium, liver and lungs; septic hyperplasia of the spleen; stasis catarrh of the esophagus, stomach and duodenum; abrasions of the skin of the anterior abdominal wall, right side. A case of brain injury with progressive signs and hemiplegia due to cortical and subcortical hemorrhages with edema simulating extradural hemorrhage."

DISCUSSION

Cases in which the typical and characteristic syndrome of extradural hemorrhage occurs are usually easily recognized and do not present a special problem. On the other hand, there is a large group of cases in which the diagnosis is extremely difficult because the extradural clot develops while the patient is unconscious from other causes.

This brings us to the principal point in the discussion, the lucid interval.

Lucid Interval. This interval is subject to the widest variations, from several days (Case IV) to complete absence (Case II). In the event that the extradural clot was the only cause of coma one would expect the lucid interval to be in proportion to the size, character and number of the bleeding vessels. Bleeding from an artery should cause the clot to form more quickly and hence the lucid interval should be shorter, whereas bleeding from the dural sinus or from the accompanying meningeal vein should occur more slowly. It seems highly improbable that the bleeding vessel, whether it be artery or vein, strips the dura from the bone. Those of us who have performed the classical operation for resection of the posterior root of the Gasserian glanglion in trigeminal neuralgia are well aware of the difficulty with which the dura is separated from the bone, even in people between the ages of twenty-five and forty-five. If for no other reason than this, one would have to accept a view similar to that advanced by Sir Charles Bell, namely that the blow separates the dura from the bone and the bleeding occurs into the space thus formed. Certainly when the clot has attained a sufficient size and in the presence of arterial bleeding, further stripping at the edges of the clot might take place. If the dura is shaken off the bone by the blow, then we expect the clot to form more rapidly in the presence of arterial than in venous bleeding and hence the time taken for the development of neurological signs and the length of the lucid interval may be some guide as to the type of bleeding to be anticipated. However, in the cases quoted here and in others that I have seen and studied, no such prediction can be made. The shaking of the dura off the bone, and its adherence to the overlying bone in the face of the advancing clot, seem to be the principal factors in the length of the lucid interval (Case IV and Case VI).

The lucid interval may be *completely absent* because of (1) severe brain injury, or (2) alcoholism. It is to be remembered that alcoholics are particularly liable to injury (Case I, arrested for drunkenness and "fell" in his cell, and Case VI) and one condition does not exclude the other. The most serious difficulty, however, occurs when the patient remains unconscious due to injury of the brain while the extradural clot is forming, and here only constant neurological observation of the patient will point to the insidious onset of a clot (Case II). The development of the clot will manifest itself by the *gradual* onset of hemiparesis and hemiplegia, and by deepening coma perhaps accompanied by dilatation of the homolateral pupil.

Hemiplegia. Even in the unconscious patient the presence of a hemiplegia can be demonstrated by firm supraorbital pressure. Supraorbital pressure, if properly applied to the supraorbital nerve, is exceedingly painful and all but the most profoundly comatose patients will respond to it. However, if there be a large extradural clot, the individual is unable to move the affected side, whereas with the sound side purposeful movements are made to remove the noxious stimulus. Certainly in the absence of hemiplegic signs the supra-orbital pressure can be continued until the investigator satisfies himself that all four extremities can be moved. This important method of demonstrating a hemiplegia in an unconscious patient is not new. I have never known it to fail and to me it is by far the most trustworthy method. Other methods of determining a hemiplegia in an unconscious patient have not been reliable in my hands. Of course, when the inability of the patient to move the affected extremities is noticed, certain corroborating signs should be found, such as the presence on the affected side of increased deep reflexes, absent abdominal reflexes and Babinski's sign. The first sign of the extradural clot may be a monoplegia later developing into a hemiplegia. The arm is always affected more than the leg.

The Pupil. In some cases the state of the pupils may be of some localizing value, as in Case II, Case III and Case VI. This brings up the question of the Hutchinson's pupil which at first is a small reacting pupil,

and later becomes a dilated and fixed pupil on the same side as the lesion. In other words, in the early stages of the formation of an extradural clot the pupil on the same side as the lesion is smaller than that on the opposite side and it reacts to light; this is supposed to be due to cortical irritation. In the later stages, the pupil becomes larger than that of the opposite side and it is not only dilated but fixed; this is presumed to be due either to paralysis of the cortex or interference with the third nerve. In Case 11 there was a divergent squint indicating to me that the external rectus on the affected side was unopposed and which I took to be due to a paralysis of the third nerve affecting the internal rectus and causing dilatation of the pupil. The position of the clot, which extended downward into the base, also made this a tenable idea. In the late stages of increasing intracranial pressure due to brain injury, both pupils become dilated and fixed and when this is so there is very little that can be expected from any form of therapy. Intraocular hemorrhage may cause a large fixed pupil.

The Bleeding Vessels. As Wood Jones has pointed out, the bleeding of extradural hemorrhage by no means always arises from a ruptured branch of the middle meningeal artery. On purely anatomical grounds the meningeal veins are more vulnerable than the arteries and this is supported by observations at operation. During the operation it is sometimes difficult to decide whether the bleeding is arterial or venous but the point can be speedily determined by having an assistant compress the common carotid artery on the affected side against the carotid tubercle. Arterial bleeding from the dura stops while the pressure is being applied, while venous bleeding would, if anything, be increased. This increase of venous bleeding is due to the fact that it is technically well nigh impossible to compress the common carotid artery without to some extent obstructing the internal jugular vein. This simple test should help the operator to decide on a course of action; whether to enlarge the craniectomy and look further for venous oozing; whether to pack the wound knowing that there is not any arterial bleeding; whether to look further for the torn artery or whether to ligate the external carotid artery in the neck knowing that this will stop the flow from an inaccessible branch of the middle meningeal artery. There are times when the condition of the patient does not warrant a prolonged search for bleeding vessels and in these cases packing the wound for venous bleeding or tying the external carotid artery for arterial bleeding seem the most practical steps to take.

It was a matter of some interest to look back and try to find out how many times the middle meningeal artery has had to be tied during the course of removal of the extradural clot. Of the 9 cases that I have seen it appears only to have been injured four times. As far as I am aware, no careful study has been made of this point. It has always been assumed that in middle meningeal hemorrhages the middle meningeal artery had been injured. Only 3 cases are reported by Wood Jones, and other authors do not discriminate between torn "meningeal vessels," torn "meningeal artery," or torn "meningeal veins."

It was discovered after a good deal of search that ligation of the external carotid was suggested by Murphy, and that he advocated it in cases which were seen early and in which symptoms were slowly progressive and in which the craniectomy might appear to be more than the patient could stand. From what we now know, it would appear that this procedure carried out on Murphy's indications would be successful in only a certain number of cases in which the bleeding happened to be arterial. Ligation of the external carotid artery has been used as an adjunct to craniectomy by Howse and by Watkins. During the Civil War resort to tying of the common carotid was frequent (Liddell 1862 and 1868). This procedure is, of course, both dangerous, on account of the possibility of hemiplegia, and unnecessary.

The fact that the middle meningeal artery or its branches is not always the source of the hemorrhage suggests that the term "middle meningeal hemorrhage" is not a good one and that the term "extradural hemorrhage" would be a better one. It is true, however, that the extradural hemorrhage is nearly always derived from either the meningeal veins or arteries, but nevertheless it is my opinion that attention has been too closely focused on the artery.

The Spinal Fluid. The presence of bloody spinal fluid in a patient suspected of having an extradural hemorrhage sometimes gives rise to confusion. This need not be the case if it be remembered that violence sufficiently great to rupture dural vessels is likely also to rupture cerebral vessels, in which case intradural and extradural bleeding would occur concomitantly. As a matter of fact, this is most frequently the case as shown both by clinical study and by study of pathological material. However, the insidious onset of coma and hemiplegia in the presence of clear spinal fluid, following a head injury, is almost certainly an extradural hemorrhage. The pressure of spinal fluid does not give much information for it probably depends largely on the accompanying edema of the brain. The pressure may be raised but it does not help one to determine the nature of the lesion causing the increased intracranial pressure.

The Injury to the Underlying Brain. It occasionally happens that the brain immediately subjacent to the meningeal vessels is contused and this leads to weakness of the opposite extremities. This condition is progressive up to a certain point and it is usually unaccompanied by marked alterations in consciousness. If any doubt exists as to the possibility of extradural hemorrhage a burr hole should be placed over the suspected motor cortex under local anesthesia.

Certainly in a large number of cases the damage to the brain is such that it masks the symptoms of the oncoming extradural hemorrhage, and even if the extradural hemorrhage is successfully recognized and treated, a satisfactory outcome is by no means certain.

The Skull Fracture. An extradural hemorrhage may occur with or without fracture of the skull, and the ultimate recovery of the patient does not depend on the damage to the skull but on the damage to the brain. It frequently happens, however, that a fracture of the skull is responsible for the tearing of the meningeal vessels at the site of the fracture. The procedure of choice is to include some portion of the fracture in the craniectomy. It is not always possible to include all of a horizontal linear fracture in the craniectomy wound.

A depressed fracture may sometimes produce signs resembling those of extradural hemorrhage, but in these cases the signs are present immediately after the injury and they are not progressive, and the *x*-ray picture is decisive. *X*-ray pictures are not always available for it may not be considered advisable to move the patient to the *x*-ray room. Under these circumstances the site of operation must be determined by the neurological signs.

DIFFERENTIAL DIAGNOSIS

The following conditions have to be distinguished from extradural hemorrhage:

1. Depressed skull fracture with brain injury;
2. Localized cerebral contusion;
3. Localized cerebral edema;
4. Subdural hematoma;
5. Cerebral hemorrhage;
6. Other nervous diseases causing hemiplegia and coma.

In all of these conditions the history of the case is of prime importance and will usually serve to guide the medical attendant toward a correct diagnosis. However, when patients arrive at hospitals having been found in the street, a history may be unobtainable and treatment may have to be instituted without delay. It is most important that the patient's state of consciousness be noted immediately and that information be obtained as to whether or not he moved all his extremities

since the supposed injury. If a history be obtained of deepening coma and hemiplegia, the indications are clear. However, an individual may have a cerebral hemorrhage, and then fall and fracture his skull; without a history he presents a picture of head injury with coma and hemiplegia. In such a case a diagnostic burr hole may have to be put in the appropriate temporal region to rule out an operable lesion. This can be done under local anesthesia and with very little risk to the patient. If an extradural clot be absent, a nick may be made in the dura and if the surface of the brain is seen the chances of a subdural clot are very remote. Contrary to general belief, a subdural clot may form fairly rapidly; I have operated on one four days after injury. By clot I do not mean the thin layer of blood over the hemispheres that accompanies most severe head injuries.

There are a few remote possibilities such as stab wounds of the temporal region, hemorrhage into a tumor, meningo-vascular syphilis, leaking cerebral abscess, which need not detain us here.

MEDICOLEGAL

Because of the possibility of a lucid interval of variable time, this subject is of medicolegal interest. An individual may be able to leave the place where he was injured, and when found may be unconscious so that no explanation of the sequence of events can be given. The behavior of the person with an oncoming extradural hemorrhage may be peculiar in that he may seem confused, even violent, and he may have taken a drink or two to fortify himself. The unintelligible mutterings of an aphasic person who smells strongly of alcohol may easily lead to a diagnosis of "alcoholic" psychosis (Case VI). His behavior may then bring him under the notice of the law with the result that he is thrown into a cell for being drunk and disorderly, only to be found next morning dead from an extradural hemorrhage. At other times a person may be found unconscious under circumstances which suggest assault and only careful investigation brings out the true state of affairs. In the Everton murder case quoted by Jacobson, a young man was struck behind the ear with a wooden roller about 5:00 P.M. At 6:30 P.M. his wife took him to the doctor's office, into which he was able to walk, but by 9:30 that night he was unconscious and he died twelve hours later of an extradural hemorrhage. The man who hit him was convicted and hanged. Two lives could have been saved by a proper diagnosis. In another case a drayman was found unconscious in a stable with a scalp wound. Grave suspicions were entertained that he had been assaulted; however, the possibility of his having fallen in a fit and of his having been kicked by a horse were taken into consideration. Eventually all the evidence of witnesses was pieced together, and it was found that he had fallen off his wagon some distance away and had climbed back into the wagon, driven home, put his horses up and had then fallen unconscious in the stable. He died of an extradural hemorrhage.

In some textbooks cases of traumatic apoplexy are described in which a delayed extradural hemorrhage is said to occur, the delay often being of a month or more. Neither Dr. Kearns of the Cook County Coroner's Office nor I have seen an extradural hemorrhage of this kind, and it seems possible that the words "extradural" and "intradural" may have been mixed. The chronic subdural hematoma, of course, might easily produce such a course.

Hughlings Jackson was well aware of the difficulty in some cases, in which the history is not clear, of differentiating cases of alcoholic intoxication from those of cerebral lesions.

For purposes of practice we have to study alcoholic intoxication carefully. It is well known that men fatally ill from cerebral lesions are sometimes locked up in police cells for drunkenness. It needs insisting on that there may be from grave intracranial lesions, soon causing death, not only insensibility, but manner and conduct like that of a person partly intoxicated by alcohol. Dr. Stephen

Mackenzie and I have recorded a fatal case of meningeal hemorrhage producing a condition which, without history of onset, would—certainly by the non-medical—have been taken for drunkenness. The patient was violent, swore, and, what is more striking, there was a purposive action; the patient got the chamber utensil from under his bed and used it. At the London Hospital we occasionally have under care men who, after sucking raw spiritis out of a cask, are seemingly as deeply comatose as other men are after large and fatal cerebral hemorrhage. Without the history of the circumstances and of the mode of onset, the diagnosis of apoplexy produced by alcohol from that produced by cerebral hemorrhage is very difficult, and may be impossible. For an hour or two after injuries to the head there may be a condition very like that of a man slightly drunk. This is an important matter. I was once consulted by letter on the case of a clergyman who was accused of drunkenness—a serious thing for a clergyman. I was able to say that the condition described might have resulted from an injury to the head that the patient had had. It is to be borne in mind that after an injury to the head a patient may act elaborately, if foolishly, whilst "unconscious"; that is, on his recovery he remembers nothing of his strange doings—a thing evidently of medicolegal importance.

Traumatic delirium is a definite clinical entity to the psychiatrist.

TREATMENT

The treatment of the condition has been indicated at various places and further discussion is unnecessary. It depends on what is found at operation. It is my custom to make a hockey stick incision in the temporal region so as to get better exposure. As a rule, the whole procedure is done under local anesthesia.

CONCLUSION

1. Many cases of extradural hemorrhage are missed because they do not present the classical syndrome.

2. Extradural hemorrhage may occur *without*

   a. Lucid interval
   b. Skull fracture
   c. Clear spinal fluid

3. The middle meningeal artery and its branches are by no means always the source of the bleeding, more often the meningeal veins or cranial venous sinuses are its source.

4. Arterial bleeding, if uncontrollable at its source, can be controlled by ligation of the external carotid artery in the neck.

5. The best indications for operative interference in a patient unconscious from brain damage are deepening coma and the insidious onset of hemiplegia.

6. In cases of doubt, a burr hole in the temporal region under local anesthesia is not dangerous to the patient and determines the presence or absence of an operable condition.

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CLINICAL OSTEOMYELITIS OF LONG BONES

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OSTEOMYELITIS is a well understood term and one usually understands its connotations. However the practice of orthopedic surgery gives the orthopedic surgeon the feeling that many general practitioners and even general surgeons fail to realize the significance of the term when applied as a diagnosis.

Four general types of bone infection will be discussed in this paper. The general subject of osteomyelitis is protean in character, each case being different from the one preceding and the one to follow, but from a clinical standpoint the group separates into several classes.

The primary class is that in which the train of events is simple, a clump of bacteria from a germinating focus of infection lodges in a terminal or near terminal blood vessel in the metaphyseal region of a long bone. The originating focus may be a boil, in which case the infecting organism is usually staphylococcus albus or aureus, or the focus may be a tonsillar crypt or adenoid, in which case the infecting organism is usually a hemolytic streptococcus. In any event the clump of organisms lodges in the metaphysis and infection occurs. Bone infections differ from soft tissue infections in that the trabeculae, the cortex and the periosteum act as definite limiting and enclosing walls causing the production of pus under pressure. This accumulated pressure in turn accounts for the fulmination with extreme toxemia and prostration which accompanies acute osteomyelitis.

Returning again to the simple class of bone infections, the following pathology may be traced. An infected embolism is lodged in the metaphysis or region just above the epiphysis. There is then the formation of an abscess within the bone presenting symptoms of some aching pain located near the joint, advancing fever, toxemia. Overnight the infection may invade the subperiosteal region and immediately there is severe pain and tenderness, redness, swelling, high fever, and marked toxemia. If the osteomyelitis is to remain within our category of simplicity it is essential that the subperiosteal abscess be drained and a window cut through the cortex deep into the spongy bone to drain the bone abscess. The necessity for speedy drainage is not so great however as to negate all surgical judgment. If the child is dehydrated, as they so often are, time should be taken to restore a normal water balance, even if this procedure takes a few hours.

The condition of the child being the best possible, the surgery should be done swiftly and thoroughly; usually the sin is on the side of omission rather than commission. The wound should be packed with vaseline gauze and the extremity immobilized as for a fracture. The packing may be left in until it is extruded by the basal granulation tissue. The outer dressings may be changed occasionally to avoid malodors. This case usually goes on to early healing, though one must expect small recurrent abscesses from time to time from which are extruded or extracted bits of dead bone or sequestrums. Crutches or slings are used until there is *x*-ray evidence of sufficient bony support for the part.

The second class of osteomyelitis is what might be termed the septic type. The invading organism is usually the hemolytic coccus either strep or staph and the picture is one of virulence and devastation. The child is pitifully sick, with high fever, dry

tissues, high white count, low red count, the picture of a septicemia, plus the signs of an acute osteomyelitis in one or several locations. In addition to the bone infection there may be suppurative processes in one or more joints.

The outlook is poor. Intravenous fluid and often transfusion are necessary preoperative measures. Surgery again should be radical for usually the infection in the medullary cavity extends far beyond any demonstrable destruction by *x*-ray or clinical examination. Multiple foci may have to be invaded and the swiftness and dexterity of the surgeon may be an important factor in the outcome of the treatment.

Fig. 1. (Case iv.) Delayed type, no treatment for eight weeks. This roentgenogram taken twelve weeks after onset. Note sequestrated shaft and heavy involucrum which has been laid down in path of subperiosteal abscess.

Fig. 2. (Case iv.) End result; clinically well. Note extensive new bone production, especially in metaphyseal region following area of old abscess.

The surgical procedure is as before, excision of an ample window in the lower diaphysis and metaphysis, vaseline pack and adequate immobilization on an already prepared splint if possible. Intravenous fluids and later small transfusions follow the surgery. Intravenous administration of organic mercurial preparations such as metaphen are often life-saving in combatting the septicemia, as is also sulfanilamide (prontylin.)

The prognosis in this septic type of osteomyelitis is poor, particularly if the invading organism is the hemolytic staphylococcus. The hemolytic streptococcus is serious but frequently is not fatal.

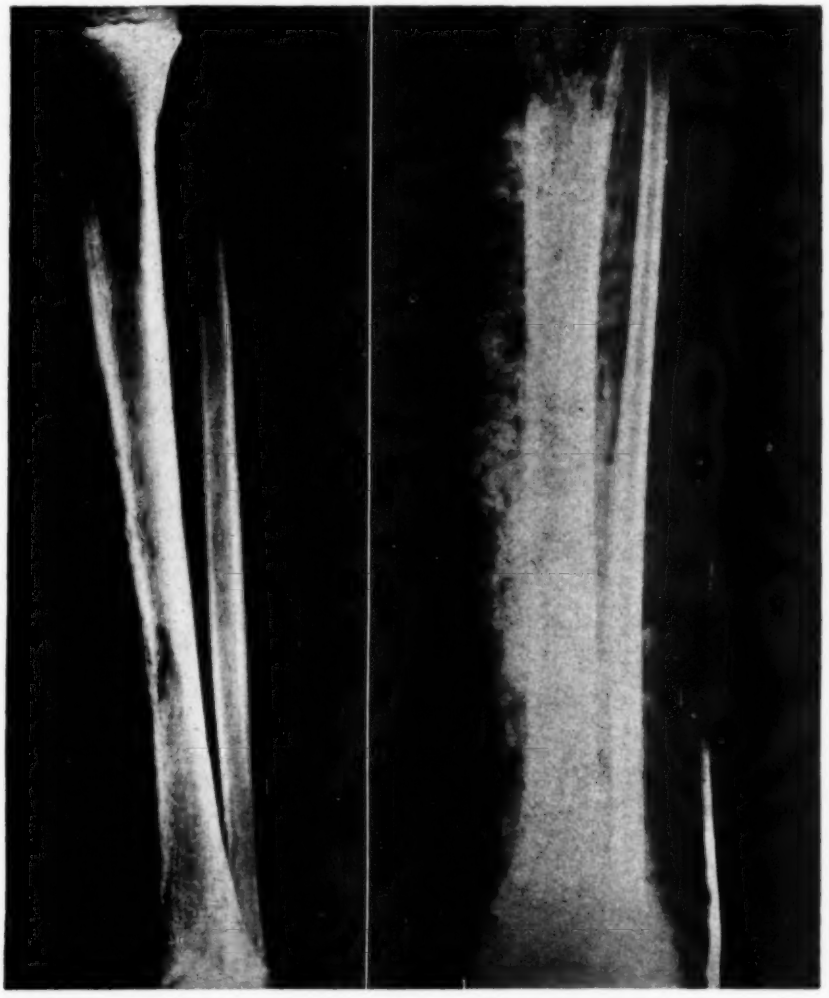

Fig. 3. (Case v.) Seventeen days after onset. Note involvement of whole shaft from lower metaphysis to tibial tubercle.

Fig. 4. (Case v.) Tibia has been guttered. Process involves whole bone. (Taken through plaster splint.)

The third class of osteomyelitis represents those late cases in which treatment has been delayed and in which there has fortunately been no fatal termination. Here we find a definite pathological picture. The extremity is swollen and edematous. There is usually a draining sinus where some well meaning colleague has "lanced a boil," actually a subperiosteal abscess which has broken through to the soft tissues. The *x*-ray picture will show a destructive metaphyseal lesion involving the whole bone structure with a central destruction throughout the length of the bone. There may be several sequestrums forming in the metaphysis, and the epiphysis itself will be moth eaten in appearance. Needless to say the epiphyseal line is irrevokably destroyed with the loss of all possibility of growth at that end of this particular long bone.

The treatment is to open the full length of the demonstrable abscess cavity, usually from the involved epiphyseal line nearly to the opposite end. If sequestrums are clearly demarked they should be removed. The bone is saucerized and the wound again

packed with vaseline gauze and the limb splinted.

This brings one to a discussion of the fourth class of bone infections, known as chronic osteomyelitis. Nearly all cases of acute osteomyelitis gravitate toward one or the other of the two types under this classification.

Fig. 5. (Case v.) Sequestration of entire shaft. This was removed four months later. (In plaster splint.)

Fig. 6. (Case vi.) Chronic osteomyelitis (ten years). Pencil of dead bone in old drill hole removed and patient well.

The simple chronic infection is one in which the window in the bone is healing slowly and from the edges and the depth of which are being extruded tiny particles of dead bone, or "bone sand" as it is called. These small pieces gradually work their way to the surface forming little abscesses either in the old incision line or wherever they may chance to point. The abscess ripens, breaks, discharges its cargo of sand, perhaps only one particle, and heals. This process may repeat itself many times during the two or three years following an acute infection, but gradually healing takes place and the limb becomes sound.

The more serious chronic infection usually follows the more serious types of onset, particularly that in which treatment has been delayed. Here one is almost certain to find one or more large sequestrums, for the curative chisel in the acute stage

leaves behind it a layer of dead bone cells which must slough out. Then too the side walls of the original cavity (the original cortex) may have been so badly involved that a whole section of the shaft may become a sequestrum. Of course as the sequestrum dies and weakens its place is taken by the surrounding involucrum or new bone so that the skeletal structure is maintained. Guard must be maintained against fractures during this period as occasionally accidents have occurred to this new bone.

The treatment consists in the removal of the dead bone with as little trauma as possible, but the time at which this treatment is instituted is of great importance. Watchful waiting should be the byword in chronic osteomyelitis for the longer one can dissuade the parents and friends from insisting upon an operation the easier it is to get the sequestrum out without new damage, which just repeats the process of destruction. The intrepid surgeon has no place in chronic bone infections.

Six case histories will be presented demonstrating the simple, the septic, the delayed and the chronic types of osteomyelitis going somewhat into detail to illustrate these classes of infection.

CASE REPORTS

Case I. (Simple) B. W., female, aged 7 years gave a history of boils immediately preceding the onset of a swelling above the right ankle five days ago. The ankle is now swollen, painful and tender just above internal malleolus, the temperature is 105°, and the white blood count shows 16,800 cells with 73 per cent polymorphonuclears. The general condition good. *X*-ray pictures show an area of rarefaction in the tibial metaphysis. Operation was done at once and a subperiosteal abscess was found. A window cut in bone, revealed an intramedullary abscess. The wound was packed with vaseline gauze. The leg was placed on a molded posterior plaster splint. She received intravenous fluids. Recovery was uneventful except for an occasional small abscess with tiny sequestrum.

Case II. (Septic) J. W., female, aged 10 years gave a history of a severe cold immediately preceding the onset. The first symptoms were pain in the right knee region, referred from the hip, then pain and swelling in the right ankle, fever of 104°, vomiting and malaise. The temperature was 105°, white blood cells 17,250, with polymorphonuclear 91 per cent, red blood cells 3,390,000, with hemoglobin 79 per cent. *X*-ray pictures showed a questionable area of rarefaction in the tibial metaphysis. The tibia was opened the day following entrance; pus was found, the wound packed and the leg splinted on an already prepared splint. Two days later, intravenous metaphen was given for a frank septicemia with a temperature 104 to 105°. The blood culture was positive for hemolytic streptococcus as were cultures taken of the wounds. A month later, the child also had an empyema of the left chest, and showed a focus in the right elbow region, right os calcis, right second lower molar region and an involvement of the right middle ear. The condyle of the right humerus was explored and drained, and the tibia guttered for a further extension of the process. A consultant advised against opening the hip region as the process appeared to be quiescent. She was sent home after a hospital stay of two and one-half months. Five months later she returned to the hospital for removal of sequestrums from the tibia and the elbow regions. The molar abscess also incised and drained. She is now up walking and living a fairly normal life. There is some limitation of motion in the elbow but the hip is quiet except for an occasional referred pain to the knee region.

Case III. (Septic) F. L., male, aged 9 years, without any known cause developed pain in his right thigh. Hot compresses were applied by mother and he was brought to the hospital after two days. His temperature was 103°, white blood cells 15,100 with 90 per cent polymorphonuclear cells. He was a very sick patient, irrational and acetone on the breath. He was transfused and operated, a large soft tissue abscess, and a large subperiosteal abscess being found. A long gutter through the cortex disclosed gray marrow, necrotic in appearance, but no free pus. The wound was packed and a spica cast was applied. Culture showed pure staphylococcus. He passed a poor night; the next day the pulse became thready and weak, the abdomen was distended, his

color cyanotic and the patient died a septic death.

Case IV. (Delayed) R. B., female, aged 5 years, had a history of bronchopneumonia followed by swelling and tenderness in the upper third of right leg. Hot compresses had been applied, the abscess had pointed, and when opened pus had been obtained. Five weeks later the pain and swelling increased and patient was brought to the hospital. The temperature was 99.5°, leucocytes 9200 with 66 per cent polymorphonuclear cells. The general condition was good. At operation a large subperiosteal abscess was found, completely surrounding the upper tibia, with some involucrum. The upper half of the tibia was guttered and packed and a splint applied. She returned home in one month. She was in an auto accident and even with the splint sustained a fracture of the shaft of the tibia through the diseased area. The involucrum had healed, however, and soon she was walking on crutches again. She is now well except for an occasional small abscess.

Case V. (Delayed) D. M., male, aged 8 years, sustained a contusion of the right ankle which became swollen, hot and tender, as did the right wrist. The diagnosis was made at once but operation was refused. Two weeks later he was sent to the hospital with a greatly swollen, hot, red and tender leg from the knee to the ankle, and a swollen right wrist. The temperature was 103°, the white blood count was 18,300 cells with 90 per cent polymorphonuclears. *X*-ray pictures showed elevation of periosteum throughout the length of the tibia, with cortical destruction through the same area, and a localized osteomyelitis of the ulna. The general condition was supported by fluids and transfusion and the tibia guttered from its upper to its lower epiphyseal lines. The ulna was also opened. Splints were applied. Blood culture showed staphylococcus albus. Repeated transfusions aided in convalescence and he was discharged from the hospital in four months with a very large sequestrum comprising the whole shaft of the tibia, partially enclosed in involucrum. In four months more the sequestrum was visible in the wound, the new bone was strong enough to support the part and, feeling satisfied nothing would be gained by further waiting, the patient was again hospitalized and the dead bone removed. Progress since that time has been uneventful save for occasional small abscesses.

Case VI. (Chronic) H. F., male, aged 28 years had a contusion of the thigh sustained in a football game in 1926. Acute osteomyelitis of the lower femur followed. He ran a high fever, and lost 40 pounds. He was operated upon by a local surgeon, the wound closing in three months. The year following, 1927, he was reoperated and several small sequestrums were removed. Subsequently several pieces of dead bone have been extruded but no marked flare-ups have occurred. Christmas Day, 1935, he played baseball after which the thigh became hot, swollen and painful. Compresses were applied but the abscess increased in size and roentgenograms showed a pencil of dead bone in an old drill hole in the femur. (The cells were killed at the time of the drilling.) The thigh was opened and a large quantity of pus, lying extraperiosteally in the soft tissues, was evacuated and the sequestrum was removed from the old drill hole. The wound was packed. In two weeks he was on crutches and to work in one month.

SUMMARY

Osteomyelitis may be grouped clinically into four classes: (1) the simple or uncomplicated; (2) the septic; (3) the delayed or destructive; and (4) the chronic.

Careful surgery, radical when required, conservative when possible, is the keynote of success.

A PRACTICAL INDIVIDUAL FILING SYSTEM

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ONE of the problems of the physician is to keep abreast of the current medical literature. Another problem is to file and classify the material read. For my own use I have devised what has proved to be a simple, practicable and efficient system. It provides for orderly classification of articles by author, subject and journal, and for abstracts of all articles arranged for easy reference. The possibility of error in references is almost completely eliminated by having them recorded in one place only. Although primarily designed for one engaged in a specialty, the system is so flexible that it can be adapted to a broader field. It is not designed for general library use, but is strictly for individual use or for use by a small group whose field is limited. I use it in the specialty of plastic and reconstructive surgery.

No filing system, however meritorious, is a substitute for reading of the literature. Any scheme requires some time and effort but its value is in direct proportion to the diligence with which one keeps it up to date.

DESCRIPTION

This system consists of a book for references (Fig. 1*a*), a book for abstracts (Fig. 1*b*) and cards for the author and subject index (Fig. 1*c*). Perhaps the most original part of the system is the small book of references, which is the only place where the name of the journal, the volume number and the inclusive paging are found. This reference book is of a standard loose-leaf type (pages 3¾ by 6¾ inches). Its contents are separated by a series of tabs which bear consecutive numbers (Fig. 1*a*). Each number represents a journal; this number is used consistently throughout the index. The page illustrated (Fig. 2) was taken from my book of references and happens to represent journal number 8, Journal of the American Medical Association. This numeral 8 is therefore known as the journal number and forms the first part of the "reference number" which will be discussed presently. In the column on the left side of the page will be noted consecutive numbering. Each of these numbers distinguishes the reference opposite it (that is, on the same line) from all others in this particular journal. This number forms the second part of any reference number. In the back of this reference book is an alphabetically arranged list of the journals which serves as an index.

For illustration, the reference number 8-13 signifies journal number 8 (Journal of the American Medical Association), and the thirteenth reference recorded. Similarly, 10-15 indicates journal number 10 and the fifteenth reference recorded. These references are recorded as soon as the article is seen, perhaps some days before it is read. When an article has been read and abstracted, a check (✓) is placed in the column on the right side of the page opposite the reference, thus signifying that this step is complete.

The book for abstracts (Figs. 1*b* and 3*a*) is also of standard loose-leaf variety (pages 5½ by 8½ inches). It likewise contains a series of numbered tabs similar to those of the reference book. Attention is called to the rectangular space in the upper right corner which contains the reference number obtained from the reference book. This sheet is filled out completely at the time the abstract is made. The small square on the upper line is used by the abstractor in evaluating the article.

By placing the letters A, B, C, or D in this square an indication of the relative importance of an article is recorded.

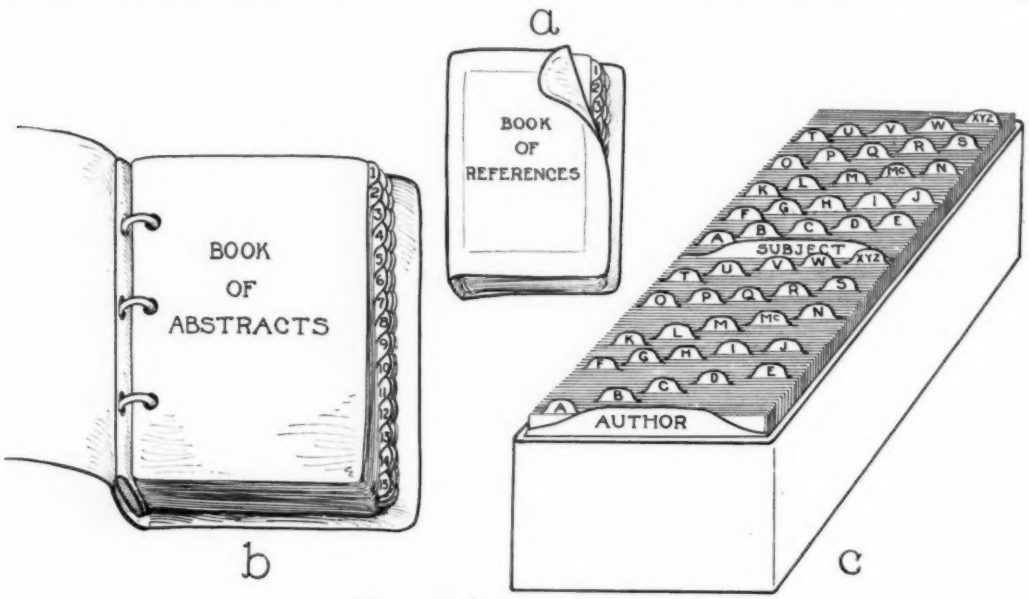


FIG. 1. Units of the system.

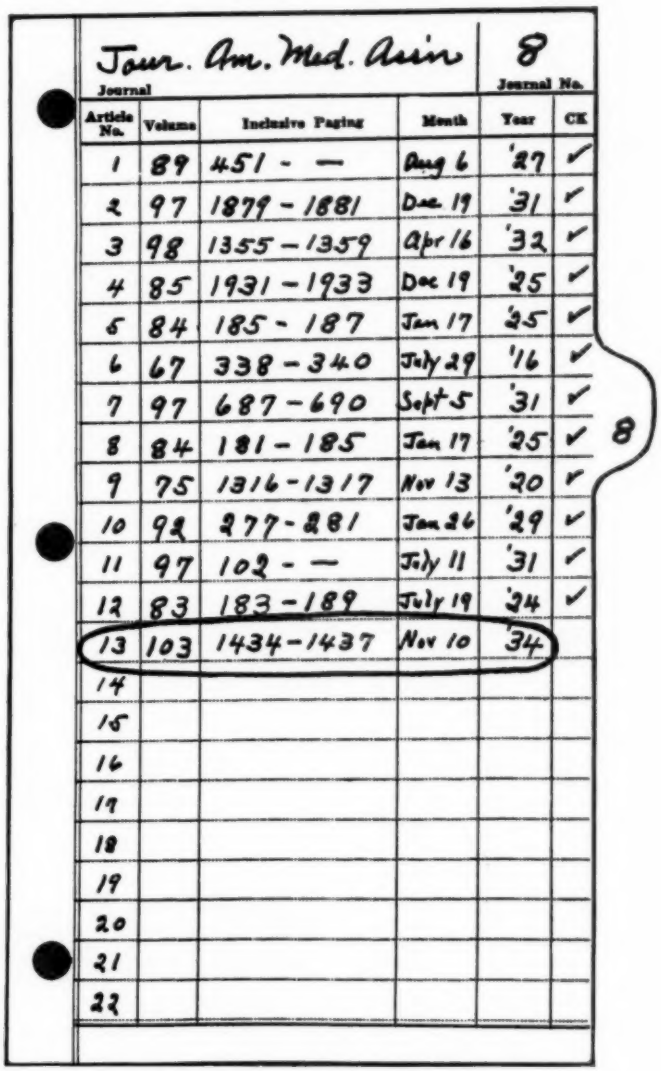
The cards for the indexing of authors and subjects are of standard size (3 by 5 inches). The author card (Figs. 3*b* and 3*c*) is blue and on it appear the name of the author, year of publication and complete title. In the space in the upper right corner is recorded the "reference number" which corresponds with that on the abstract sheet and which has been obtained from the book of references. The cards are filed alphabetically in the author index. When a writer has published several articles, necessitating as many cards, they are further arranged according to year, the latest article being placed in front. The subject card is white and contains the subject, year, author's name, reference number and title of article (Figs. 3*d* and 3*e*). These cards are filed alphabetically in the subject index. When several adjacent cards carry identical subject references, they are further arranged according to year, and as a further subdivision according to author.

HOW TO PLACE AN ARTICLE IN THE SYSTEM

Suppose, for example, the following article is selected: "Use of reconstructive surgery in certain types of deformities of the face" by G. B. New and F. A. Figi. It is found in the Journal of the American Medical Association, volume 103, pages 1434–1437 (November 10) 1934. The first step (Fig. 2) is to copy the volume number, inclusive paging, month and year of publication in the reference book. These data are placed on the first unused line on the page which has "Journal of the American Medical Association" as its heading. This automatically gives the article a reference number. In my index, "Journal of the American Medical Association" happens to be journal number 8, as has been said. The reference to the article has been placed on the thirteenth line and its reference number has become 8-13. Throughout all the system this article is now referred to as 8-13. When an article is discovered in a journal which is not represented in the reference book, its name is written on the next unused page. This automatically assigns a number to the journal.

The second step consists of filling out the abstract sheet (Fig. 3*a*). The reference number (8-13) is first written in its special place in the upper right corner of the sheet. The authors' names and the complete title of the article are likewise written in the spaces provided. The article is ab-

stracted; at the end of the abstract a notation is made of those subjects under which the article is to be cross indexed. process. The typewritten cards present a neater appearance and are more easily read.



Journal: Jour. Am. Med. Assn. Journal No.: 8

Article No.	Volume	Inclusive Paging	Month	Year	CK
1	89	451 - —	Aug 6	'27	✓
2	97	1879 - 1881	Dec 19	'31	✓
3	98	1355 - 1359	Apr 16	'32	✓
4	85	1931 - 1933	Dec 19	'25	✓
5	84	185 - 187	Jan 17	'25	✓
6	67	338 - 340	July 29	'16	✓
7	97	687 - 690	Sept 5	'31	✓
8	84	181 - 185	Jan 17	'25	✓ 8
9	75	1316 - 1317	Nov 13	'20	✓
10	92	277 - 281	Jan 26	'29	✓
11	97	102 - —	July 11	'31	✓
12	83	183 - 189	July 19	'34	✓
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Fig. 2. Page from book of references.

A rating is given and this second step is finished.

The last step consists of making out and filing the author and subject index cards (Fig. 3*b*, *c*, *d* and *e*). The information for these cards is obtained from the abstract sheet (Fig. 3*a*). When these cards have been made out, a check (✓) is placed opposite the notations at the end of the abstract and this last step is completed. It is to be noted that this final step of making out and filing of the author and subject cards can be entrusted to an assistant as it is purely a secretarial

SALIENT FEATURES

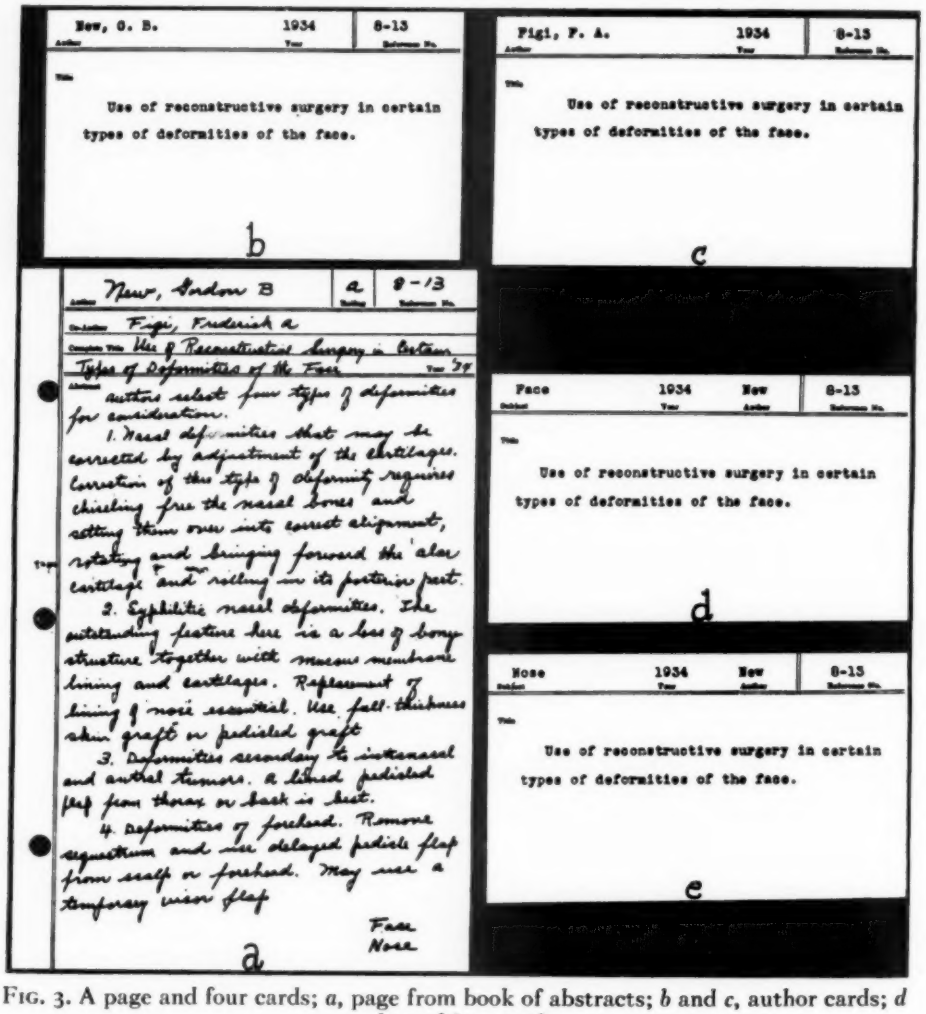
The system is readily expansible to meet the requirements of an ever increasing literature. When the book of abstracts outgrows the single volume, part of its contents is placed in a second or third volume without disturbing the continuity.

In making up a bibliography an author needs only to supply his secretary with a list of reference numbers. With these she can compile a complete bibliography from reference book and abstract sheets. Furthermore, these references are necessarily

correct because they were used originally in finding the article.

Each abstract is written on a separate sheet. If for any reason the abstract needs to be revised the corrected sheet is readily substituted. If the single sheet does not provide sufficient space for a long abstract, additional sheets can be used. Keeping of the abstracts in a book is advantageous because they are held securely together, are easily carried, remain in their proper places and there is little chance of misplacement or loss, such as may be the case with cards. For those, however, who prefer cards these loose-leaf sheets lend themselves admirably to the purpose.

By rating each article as it is read, at any time, the physician can quickly select from his files those contributions of greatest value. This feature saves valuable time, as articles of minor importance are automatically eliminated from further consideration.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Use of reconstructive surgery in certain types of deformities of the face.
b
Figi, F. A. 1934 8-13
Use of reconstructive surgery in certain types of deformities of the face.
c
New, Gordon B a 8-13
Figi, Frederick A
Use of Reconstructive Surgery in Certain Types of Deformities of the Face '34
authors select four types of deformities for consideration.
1. Nasal deformities that may be corrected by adjustment of the cartilages. Correction of this type of deformity requires chiseling free the nasal bones and setting them over into correct alignment, rotating and bringing forward the alar cartilage and rolling in its posterior part.
2. Syphilitic nasal deformities. The outstanding feature here is a loss of bony structure together with mucous membrane lining and cartilages. Replacement of lining is most essential. Use full-thickness skin graft or pedicled graft.
3. Deformities secondary to intranasal and antral tumors. A lined pedicled flap from thorax or back is best.
4. Deformities of forehead. Remove cicatrix and use delayed pedicle flap from scalp or forehead. May use a temporary vein flap
Face
Nose
a
Face 1934 New 8-13
Use of reconstructive surgery in certain types of deformities of the face.
d
Nose 1934 New 8-13
Use of reconstructive surgery in certain types of deformities of the face.
e

FIG. 3. A page and four cards; *a*, page from book of abstracts; *b* and *c*, author cards; *d* and *e*, subject cards.

Author and subject cards need never be removed from their respective files, thus eliminating the possibility of loss or misplacement. When it is necessary to refer to an article, only the reference number is required. Several such numbers may be written in the time required to write one complete reference in the ordinary manner and with less likelihood of error.

Occasionally one tries to recall the name of an article read perhaps a long time previously. With this system, it can be found readily if any one of three things about the article is recalled: (1) the name of the author or any one of the co-authors, (2) the subject about which

the article was written, or (3) the name of the journal in which it was published. If the physician recalls the author's name, he consults the author index. If he remembers the subject about which the article was written, he consults the subject index. Here the article can be located under any one of several headings because of the cross references. If the physician recalls only the journal in which the article was published, he needs only to glance through the group of abstracts under the journal in question.

One may desire to know what recent contributions a certain author has made to the literature. In this case one consults the author index, where all the articles are arranged according to year in which they were published, the most recent articles being placed in front.

If a physician is writing an article on some phase of his specialty and wishes to acquaint himself with the literature, he can refer to the subject index. Referring to my file for illustration, one might wish to know what recent contributions have been made on surgery of the lip. By referring to "lip," in the subject index, a series of cards is found, each referring to some phase of surgery of the lip. They are further arranged according to year of publication, the most recent articles in front. Since a résumé of each article is available in the book of abstracts, and because each has been rated, those contributions of greatest value are easily selected.

CONCLUSIONS

It can readily be seen that this system has numerous advantages and possibilities, but it must be emphasized again that no filing system can be used in lieu of the actual reading of the literature. I have used this system during the past year and have found it to be most satisfactory.

The printed forms herein described may be obtained from the Whiting Press, Rochester, Minnesota.

CASE REPORTS

THE MENOPAUSAL STATE*

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NEW YORK

CESSATION of menstruation occurring during the middle life of every woman is generally known as the menopause or "change of life," is probably the most pronounced subjective observation, other general signs supervene that render the majority of women undergoing this stage of their sexual career, subjects who require definite medical aid to tide them over this so-called critical or transitional period.

The causal factor producing this condition is primarily a gradual physiological atrophic process that the ovary undergoes, which at first limits and later completely prevents the development and maturation of the Graafian follicle. Therefore, ovulation does not occur and the formation of the corpus luteum is absent.

The normal menstrual cycle is dependent upon the female sex hormone, (estrin) that is manufactured in the developing and maturing Graafian follicle, as well as the corpus luteum. Therefore, this gradual atrophy limits the estrogenic stimulation resulting in irregularity of the menstrual periodicity; and while the periods may show an absence of, or increased interval varying between two to three months, as the most prominent symptom, other signs, such as flushes, sweats, irritability, etc., soon occur that take precedence in importance over the menstrual irregularity. These latter phenomena are due to the close interrelationship of the hormonal balance of the pituitary, thyroid and adrenal glands with the autonomic nervous system, which becomes affected by the diminution and later absence of estrin in the circulation.

The ovarian changes are produced by an increase in connective tissue and a diminution of the follicular apparatus. Maturing of the follicle is thereby gradually prevented in some instances, while in others a thickening of the ovarian tunica prevents rupture and ovulation, and eventuates in a cystic condition or atrophy of the follicle. Formation of the corpus luteum does not then occur and upon withdrawal of its inhibiting hormone, progestin, excessive uterine bleeding may be a prominent symptom of this transitional period in women's lives.

The onset of the climacteric has been estimated by various observers to occur from forty-one to forty-nine years. It has been generally observed that when menstruation began at an earlier age than usual the menopause was likely to be late in appearing. My own opinion in contacting numerous menopausal women, would lead me to believe that forty-seven years is a good average if one bases the initiation of this state upon the menstrual irregularities, such as the lengthening of the intervening interval. In a great many instances however, preceding interference with menstruation, there exists what can be termed a pre-menopausal stage manifested by neurocirculatory symptoms of mental irritability, mild flushes, digestive disturb-

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ances, and precordial or pseudo-anginal pains. Increased sex desire and libido may also be present as a preclimacteric symptom. These phenomena apply not only to the natural but to the surgical menopause as well.

The removal of the uterus and ovaries by operation or from irradiation, as a rule, precipitates the menopause in a short time, although in some instances, among women in active sexual life, the symptoms do not appear for several months following operation.

The symptoms of ovarian deficiency is a familiar picture. The premenstrual state described is, as a rule a precursor of the amenorrhea that follows. Concomitant with the amenorrhea and the menopausal syndrome, changes in the external as well as the internal generative organs take place. Replacement of elastic fibers with connective tissue causes a shrinkage of the labia; the vaginal mucous membrane shows fewer rugae and an atrophy of the mucous membrane produces a thin layer of epithelium that frequently becomes excoriated by desquamation, resulting in the so-called senile vaginitis, so often encountered. The cervix undergoes a similar involution, becoming smaller and harder with a diminution of its glandular epithelium and consequent lessening of secretion, unless there is a pre-existing chronic infection. The uterus gradually becomes smaller, as the atrophic changes progress, with substitution of fibrous tissue at the expense of the muscular elements. During this process there often are local symptoms of prurutis and burning, and frequency of micturition. The ovaries become fibrous and the follicles which may continue for some time do not mature and rupture, therefore ovulation does not take place and corpora lutea are not formed.

The neurovascular symptoms described as premenopausal in character are all increased in severity with full establishment of the menopausal state. Flushes and sweats may be very frequent both during the day and night and be the most predominant symptoms, which cause an insomnia of a severe type. This loss of sleep and an imbalance of the autonomic nervous system, together with psychic disturbances, may produce an extremely deplorable mental and physical condition.

The neurological symptoms may be those of vertigo, headache, parasthesia and insomnia, in the moderately average cases. Psychic disturbances no doubt occur in the majority of menopausal conditions to a mild degree, but among others who had previously possessed an unstable mental balance these disturbances may assume more severe characteristics in the form of functional psychoses or involutional melancholia. The accompanying delusions and threats and attempts at suicide in these severe types may necessarily lead to either very close and constant home supervision or commitment to an institution. Other nervous manifestations are those of vertigo, headaches, fatigue, visual and gastrointestinal disturbances, as well as mental irritability and unreasonableness and exaggeration concerning trifling details.

Other occasional associated symptoms of the menopause are periodic migraine attacks varying from mild headaches to severe typical explosions of visual disturbances, nausea and vomiting of several days duration, requiring rest in bed for the period of the attack. Painful joints are also occasional symptoms, but a true arthritis does not exist and the subjective symptoms generally disappear upon the institution of proper menopausal therapy.

The lack of control by the proper balance of hormones from the ovary, anterior pituitary, thyroid and adrenals is a causative factor in the physical and mental changes occurring during this stage. The lessening and final withdrawal of estrin releases the inhibiting influence upon the anterior pituitary lobe which allows for an increase into the circulation of the gonadotropic hormones. No doubt, there is also an increase in the thyrotropic and adrenotropic hormones, causing an

overactivity of the thyroid and adrenal glands which through the sympathetic nervous system produces the various symptoms already related.

With an overproduction of the thyrotropic hormone from the prepituitary, stimulation of the thyroid gland may take place. No doubt symptoms of apprehension, increased pulse rate, loss of weight, and increased basal metabolism, can be explained by a hyperthyroid state in a small percentage of patients undergoing the menopause.

Through the adrenotropic hormone from the prepituitary gland, stimulation of the adrenal medulla and cortex occur. An increased production of adrenalin therefore is released into the circulating blood which acts upon the sympathic nervous system causing the vasomotor symptoms of flushes and sweats. Through stimulation of the adrenal cortex, and its consequent hyperfunction, is the reason why some women assume masculine characteristics, such as change of voice and development of facial hair.

Studies made by various investigators have shown that during the menopause there is in both blood and urine determinations, a decrease in the estrogenic and an increase in the gonadotropic factor. In the latter the luteinizing factor is predominant. It is interesting to note that all patients passing through the menopause do not show these findings, that the estrin content continues to be present. This may be due to the stimulus imparted to the follicle by the gonadotropic hormone increase. The follicle however does not mature and rupture but estrin continues to be manufactured to some degree. Investigations by Goldberger have demonstrated that there are other sources of the female sex hormone than the ovarian follicle, one of which is food normally ingested which he estimated to contain 75 to 100 M. U. daily.

In the treatment of these conditions it seems practical that the introduction into the circulation of estrin in sufficient quantities to produce an inhibitory action upon the anterior lobe of the pituitary gland is the treatment of choice. Some years ago I was quite impressed with the results obtained from the administration of placenta and anterior pituitary extracts to menopausal patients, but the last few years, following the manufacture of estrogenic substances in concentrated form and accurately standardized in animal units, the exclusive use of this form of therapy has given superior results.

The clinical application of replacement estrin therapy naturally depends upon the severity of the symptoms. An initial dose of 30,000 international units of estrin (endofollicolina) administered intramuscularly will in the majority of instances relieve many of the severe symptoms within forty-eight hours. Repetition of this dosage every other day until 150,000 I. U. have been given as a rule will control the flushes, restlessness, palpitation, etc. This replacement therapy however is not of lasting duration even though relief is experienced and this fact should be impressed upon the patient, as return of the symptom complex is bound to occur, when the supply of estrin in the circulation is eventually exhausted through its excretion in the urine and feces.

Therefore continual therapy is necessary to maintain the proper inhibitory hormonal balance. This is best accomplished by subsequent injections of 30,000 I. U. once weekly. Subsequently over a period of months intermittent therapy can be tried by either reducing the number of units administered or continue the larger doses at longer intervals. To continue this replacement therapy, when a proper balance has been obtained, oral administration of endofollicolina tablets, containing 750 I. U., can be given three times daily with splendid results. However, through emotional or physical disturbances, recurrences take place, requiring occasional return to the larger dosages by injections.

Other therapeutic aids are the use of thyroid substance in those patients who

have a low basal metabolism rate. For the obesity that frequently accompanies this period of life appropriate dietary advice can be given. General hygienic measures such as fresh air and exercises and avoidance of unpleasant environment should be impressed upon the patient.

CASE HISTORIES

A few case histories are here related to illustrate the different groups manifesting the typical symptoms with their response to treatment.

Menopause with Marked Psychotic Symptoms. Mrs. J., aged forty-five years; menstruation began at eleven years, was present every twenty-eight days and lasted five days. For the previous ten months, her periods were irregular, and she had symptoms of numerous hot flushes day and night, sweats, insomnia, severe headaches, and marked physical and mental depression to such a degree that suicidal threats were frequently made. Evidently the psychic state was one bordering upon melancholia. Replacement therapy was immediately administered; 30,000 I. U. of endofollicolina were given by intramuscular injection, for three injections, with complete and immediate disappearance of all symptoms. Therapy was continued with one injection a week, for one month which controlled any recurrences. Later she was placed on oral therapy, with satisfactory results.

Average Menopause. Miss L., aged forty-seven years; menstruation began at thirteen years, occurred every twenty-eight days and lasted five days.

Premenopausal Symptoms. For the past six months has had irregular periods, misses a period, then flows twice in a month; complains of headaches, dizziness and flushes, with sweats to a moderate degree. She was placed on placenta and anterior-pituitary substance orally and her periods became more regular with reduction of the symptoms. Owing to a lowered basal metabolic rate and an increase in weight, thyroid grains ½ was given three times a day, followed by marked improvement. As the menopause advanced and menstruation a year later became more infrequent, estrin replacement in this form became insufficient as all symptoms recurred with greater severity. The patient was then placed upon large doses of estrin 30,000 I. U. (endofollicolina) administered by intramuscular injection every other day for five doses, with entire control of her symptoms. She continues to have these injections once weekly.

Surgical Menopause. Miss S., forty-one years old; menstruation began at fifteen years, occurring every twenty-eight days and lasted four days. *X*-ray menopause of two years duration, with many flushes, cold sweats, mental confusion and irritability. She was placed on placenta and anterior pituitary injections three times weekly. Continuation of this therapy for eight months gave considerable reduction of symptoms. Replacement therapy, however, seemed insufficient so she was later given 30,000 I. U. of endofollicolina by intramuscular injection once a week, with an entire disappearance of symptoms.

CONCLUSIONS

It is quite evident that in this series of 55 patients, from the clinic and private practice, exhibiting the characteristic syndrome of the menopausal state, that irrespective of the severity of the symptoms their control can be established by administration of estrin in sufficiently large doses. This control is produced by supplying to the circulating blood a quantity of estrin that will inhibit the prepituitary body from manufacturing an over-abundance of the gonadotropic, thyrotropic and adrenotropic hormones, thus controlling the glandular output, so that a hormonal balance will result. Lack of response to this form of therapy usually signifies that either insufficient units have been given or that other organic cardiovascular or renal conditions exist. Careful observations and study of each individual case, however, with supplemental glandular therapy as the needs of the patient's require will result in greatly modifying the severity of the symptoms, and render comfort if not a complete cure.

I am indebted to Dr. Sante Piracci of the Italian Drugs Importing Company for his kind cooperation in supplying the clinic with an unlimited amount of endofollicolina in both ampoul and tablet form for this investigation.

TRAUMATIC SEPARATION OF EPIPHYSIS OF LOWER END OF FEMUR

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SEPARATION of the epiphysis of the lower end of the femur is a rare occurrence. A review of the literature reveals but forty-six references to this condition since 1884. Mahorner and Bradborn in 1933, reviewed 308 cases of fractures of the femur, and mentioned the possibility of its occurrence, but had no cases in their series.

Kurlander in 1931, also reported this as a rare type of injury. He cited three cases, one of which was reduced by manipulation, another, an old injury, was reduced by open operation, and in the third amputation was necessary because of gangrene.

In 1933 Ireland was able to collect from 1911 to 1931, twenty-six cases of epiphyseal separations, of which only two were lower femur cases.

Hutchinson and Barnard in 1898 collected thirty cases of which six resulted in a useful limb. At that time they advocated the use of the fully flexed position, and showed it was difficult and sometimes impossible to reduce in the extended position. This fact is very graphically shown in the *x*-ray plates accompanying our case.

FIG. 1. Supracondylar fracture (epiphyseal separation) of the lower end of the femur with anterior displacement of the condyles.

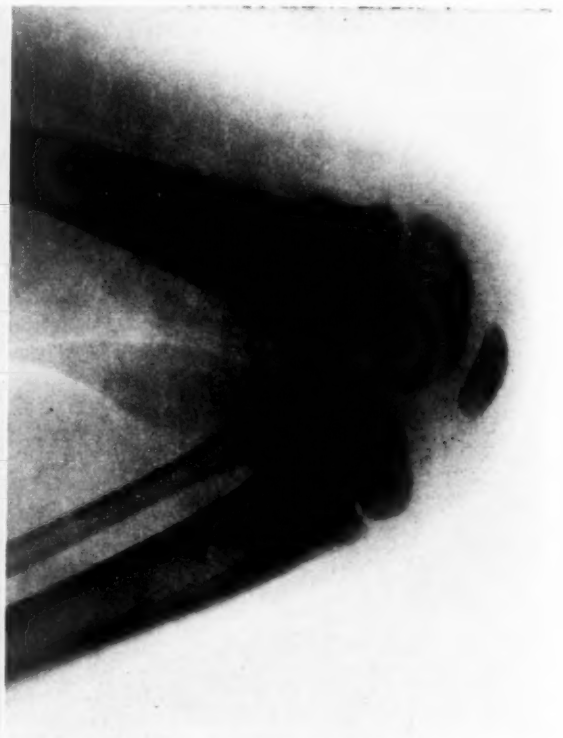

FIG. 2. Supracondylar fracture of the femur, reduced and in excellent position.

Plummer in 1932 also brought out the fact that reduction is easily accomplished by flexion, and upon extension of the leg dislocation results.

Poland in 1898 collected 114 cases. Henschen, in 1909, brought the total to 200 cases, and Russell, in 1912, added eight more. At this time he stressed the fact that

Fig. 3. Marked displacement of fragments resulting from slightly extending the knee.

Fig. 4. Reduction again accomplished by merely acutely flexing the knee.

Fig. 5. About six weeks following reduction; fracture in excellent position with evidence of healing.

Fig. 6. Fig. 7.

Figs. 6 and 7. About six months after reduction; complete extension of the knee under anesthesia. The epiphyses are in normal relationship to the bone.

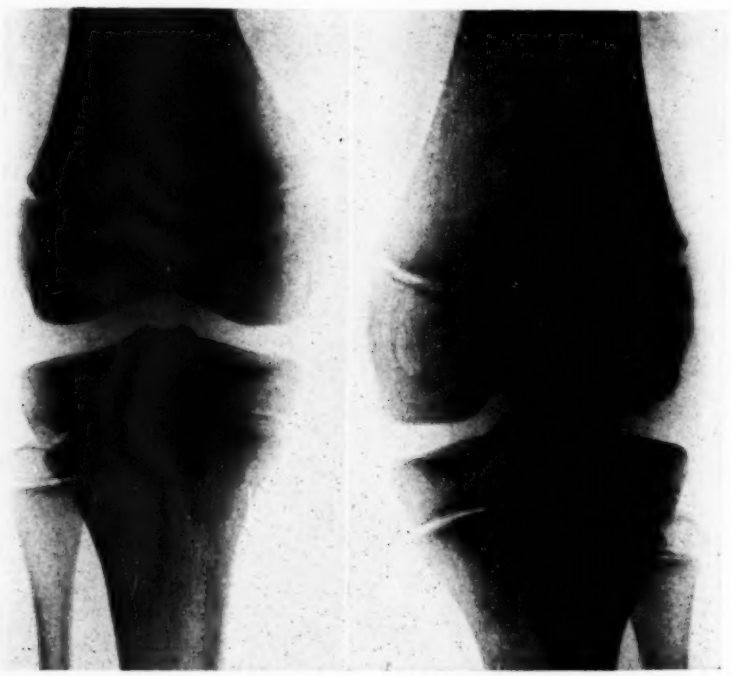

Fig. 8. About four years after injury, both thighs were placed in the same position. This *x*-ray picture shows the actual lengthening of about one inch in the injured side (left), and the enlargement of the epiphysis on the same side.

these injuries were thought to be dislocated knees, which error also occurred in our case.

Cottam in 1905 reported one case showing interference with epiphyseal growth which resulted in the development of knock knee. This occurred in the case presented.

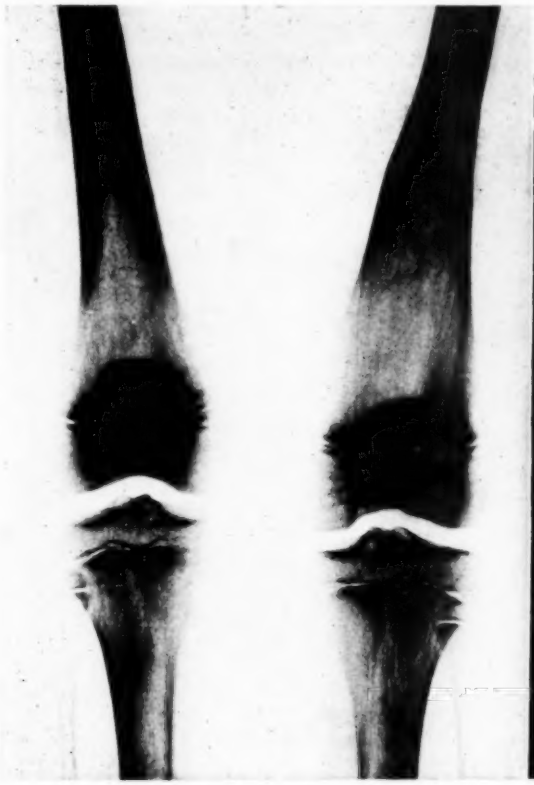

Fig. 9. *X*-ray plate about four years after injury. The actual lengthening can be measured on this plate as the upper epiphysis of the femurs are visible, and may be used as landmarks (this plate is slightly reduced in size).

Fig. 10. Photograph showing the marked prominence of the internal condyle of the left femur (knock knee) taken November 30, 1936.

Cotton (1924 edition) saw one case of increased growth, mainly on the inner side, with a total increase in the length of the injured limb amounting to one inch in three years. This is the only reference found citing the lengthening of the limb following this type of injury. The case reported below also shows this lengthening. This observation may be due to the fact that Cotton's case was observed three years after injury, and the present case was observed four years after injury, while the other cases covered in the bibliography were observed for rarely more than a year.

CASE REPORT

A white, Italian boy, aged 8 years, was brought to the hospital on August 16, 1932, at about 8:10 o'clock P.M., with a history of being struck by an automobile shortly before admission. The history indicated a direct blow to the left knee by the front wheel of the auto.

There was a deformity of the left knee. No *x*-ray being available, it was clinically diagnosed as a dislocated knee, and an immediate attempt at reduction (9 P.M.) under ether was made and a plaster cast applied.

X-ray picture taken the next day showed a "supracondylar fracture, (epiphyseal separation) of the lower end of the femur with anterior displacement of the condyles" (Fig. 1).

Another attempt was made at 4:30 P.M. to reduce the deformity under ether anesthesia.

X-ray examination now showed, "epiphyseal separation of the lower end of the femur with fracture above the condyles. There is no improvement in the position."

Due to the general condition of the patient, and the marked reaction of the tissues in the area of the injury, it was thought best not to attempt any further manipulation at this time.

On August 27, 1932, or ten days later, under fluoroscopic control, the knee was acutely flexed and the deformity was spontaneously reduced. No traction of any kind was used. The *x*-ray picture now showed a "supracondylar fracture of the femur, reduced and in excellent position" (Fig. 2). The knee was now brought out about 15°, because it was thought that too acute an angle might interfere with the circulation through the popliteal space. A plaster cast was applied and an *x*-ray plate taken, which showed a "marked displacement of the fragments" (Fig. 3). The cast was immediately removed, the knee reflexed the extra 15°, and a new cast applied. *X*-ray examination again showed "the fracture reduced and in excellent position." (Fig. 4.)

The patient was discharged from the hospital on August 31, 1932, four days after reduction, with the left knee in a cast in acute flexion.

On October 14, 1932, seven weeks after reduction, the patient was readmitted. The cast was removed, the knee extended to about 90°, and a new cast applied. This was accomplished without any anesthesia. *X*-ray report following this showed "fracture in excellent position with evidence of healing" (Fig. 5).

This cast was allowed to remain about ten days, and then was removed. Active and passive motion with physiotherapy was instituted, and gradually motion was resumed in the knee. However, complete extension was impossible, so that on March 10, 1933, six months after reduction, a knee stretching was done under ether anesthesia. At this time the knee was hyperextended and put in a plaster cast. An *x*-ray examination at this time showed, "the epiphyses are in normal relation to the shaft of the bone." (Figs. 6 and 7.)

The cast was removed at the end of five days and physiotherapy measures again instituted.

Re-examination of the patient four years after the injury on June 6, 1936, now aged 12 years, revealed that the injured extremity was one inch longer than the uninjured limb. It also showed a thickening of the entire epiphysis, but more marked on the inner side. This is revealed upon inspection by a knock knee (Figs. 8, 9 and 10).

Measurement from the anterior iliac spine to the internal malleolus on the left leg was 30 inches, and on the right leg, 29 inches.

There was also a thickening of the knee, the left measuring 11 inches above, 12½ inches at the patella, and 10 inches immediately below the knee; while the right measured 11 inches above, 11½ inches at the middle, and 10 inches below the knee.

There was marked crepitus in the left knee but no limitation of motion, no pain or discomfort. There was a slight tilting of the pelvis due to the lengthening.

SUMMARY

A case of traumatic separation of the lower epiphysis of the femur is presented. This is of particular interest because:

1. An exhaustive survey of the literature reveals its rare occurrence.
2. It illustrates the need of *x*-ray examination to make an accurate diagnosis of this injury.
3. It graphically illustrates the ease with which the condition may be reduced.
4. It illustrates the development of knock knee due to irritation of the epiphysis.
5. It illustrates the lengthening that may occur following repair of an epiphyseal injury.
6. This case has been followed for a period of four years.

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AN OVARIAN HYPERNEPHROMA*

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IT is not in the province of this report to discuss the nomenclature or origin of hypernephromas. Suffice it to say, that a hypernephroma is a peculiar and morphologically characteristic type of tumor, different from any other form of neoplasm, and was given this name by Birch-Hirschfield. Some are of the opinion that it rises from rests or remnants of adrenal cortex which have been included in the kidney (Grawitz, 1883) while others think it is derived from the renal tubules.

The degree of malignancy varies from relatively benign to highly malignant growths. There is a special tendency to the invasion of veins, and the tumor may grow into the renal vein and even into the inferior vena cava. The most frequent regions of metastasis are the bones, lungs, regional lymphatics, liver and skin.

In the literature to date there has appeared only nineteen cases of hypernephroma of the ovary. Most of these were growing from adrenal tissue at the hilum of the ovary and later involved the ovary by contiguity: only one case (Table 1) was a true metastatic hypernephroma of the ovary.

The case herein presented is a metastatic hypernephroma of the ovary occurring nineteen months after the extirpation of the original tumor which occurred in the left kidney.

CASE REPORT

Mrs. L. S., white female, aged 39 years, was admitted to Charity Hospital, September 7, 1933. At that time she complained of a rapidly growing, painless mass in the left upper quadrant of the abdomen. She first noticed, four months prior to admission, a small lump, and in three weeks it had increased to its present size. There were no symptoms referable to any of the systems, except a slight loss of weight, her weight was 112 pounds, although she had never weighed more than 120 pounds for the past eight years.

She was a multipara having had two children who were living and well at the ages of 7 and 4 years. She began menstruating at the age of 14 years and the menses recurred every twenty-eight days, lasting four to five days. There had never been any serious illnesses in her life and she had had no operations. Her family history did not indicate any familial diseases.

The physical examination did not reveal any pathological conditions, except for a moderate degree of myopia and a large hard tumor the size of a grapefruit in the left upper quadrant of the abdomen; the tumor moved only slightly with respiration. Her temperature was 98° to 99°; pulse rate 80 to 85, respirations 20 to 22. The blood pressure was 120/80.

Clinical laboratory findings were: Blood chemistry: total non-protein nitrogen 30 mg. per 100 c.c. of blood; urea nitrogen 15 mg.; creatinine 1.1 mg.; blood sugar 100 mg.

The blood Wassermann reaction was negative.

The icterus index was 3.

Fragility test (red blood cells) hemolysis began at 0.42 per cent saline and was complete at 0.30 per cent saline; the control hemolysis began at 0.42 per cent saline and was complete at 0.32 per cent.

Blood Count: Red blood cells 4,005,000 per 1 cm. of blood, hemoglobin 85 per cent (Sahli and Dare); white blood cells 6,250; with small lymphocytes 25 per cent; neutrophiles 72 per cent and basophiles 2 per cent; platelets 186,000; bleeding time 2½ minutes; coagulatime 1½ minutes.

Urinalysis: Urine acid, specific gravity 1.018; examination for sugar, acetone, diacetic acid

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TABLE I

CASES OF HYPERNEPHROMAS OF THE OVARY REPORTED IN LITERATURE

Number	Age	Number of Pregnancies	Menstrual History	Treatment	Clinical Pathological Findings	Result	Author	Date	Source of Ovarian Tumor
1	45	4	Menstrual irregularity	Operation	Tumor of the right ovary	Recovery, time of observation not stated	Peham	1899	Original
2	50	.....	.....	Operation	Large tumor of the right ovary and small tumor of the stomach	Died 3 months after operation	Peham and Paultauf	1899	Original
3	51	.....	.....	Operation	Large tumor of the right ovary	Died 2 years after operation	Pick	1901	Original
4	17	Nullipara	Amenorrhea for several months	.....	Hypernephroma of the ovary, broad ligament on same side	.....	Sternberg	1906	Origin undetermined; probable extension from broad ligament
5	53	.....	Metrorrhagia last 6 years	Operation	Hypernephroma of right ovary and left kidney; metastatic uterine fibroids	Died of shock after operation	Santi	1906	Metastatic
6	40	Multipara (3 children)	Amenorrhea	Operation	Bilateral ovarian tumors	Died 9 months after operation	Scudder	1907	Original
7	4	.....	Virilism; menorrhagia and metrorrhagia	Operation	Tumor of the left ovary	.....	Gaudier	1908	Original
8	49	Nullipara	Amenorrhea (menopause ?); did not menstruate for 8 years	Operated twice (11 months interval)	Hypernephroma of the left broad ligament (?). No trace of ovary on this side was found; ovarian tumor(?)	Well 2 years after operation	Gardner and MacCleary	1908	Origin undetermined, probable extension from broad ligament
9	....	.....	.....	.....	Tumor of the ovary; metastatis in the humerus	Died, time not stated	Meyer	1908	Original
10	....	.....	No literature available	.....	Tumor of the ovary	.....	Pfannenstiel	1908	No data available
11	27	1	.....	Operation	Tumor both ovaries	Died soon after operation with metastases in spinal column	Rosthorn	1909	Original
12	27	1	.....	Operation	Tumor of the right ovary	No information	Vonwiller	1911	Original
13	56	.....	Metrorrhagia	Operation	Tumor of the broad ligament; tumor of the ovary (uterine fibroid)	Died after ?	Alamartine and Maurizot	1912	Origin undetermined (probable extension)
14	26	Nullipara	.....	Operation	Tumor of the ovary	Well after 4 years	Levy de Pan	1924 (1920)	Original
15	3½	.....	Virilism	Operation	Tumor of the right ovary	Well after 2 years	Downes & Knox	1924	Original

TABLE I (*Continued*)

Number	Age	Number of Pregnancies	Menstrual History	Treatment	Clinical Pathological Findings	Result	Author	Date	Source of Ovarian Tumor
16	7	Nullipara	Virilism; menses at 7 years	Operation	Large right ovarian tumor attached to broad ligament	Died	Duffour & Lucien	1925	Original
17	31	Multipara	Virilism; colloid degeneration of thyroid	Operation	Tumor of the right ovary; developed in the right broad ligament and invaded ovary through hilum	Died 3 months after operation	Orru & Volpe	1926	Extension
18	30	Multipara	Amenorrhea	Operation	Tumor of the ovary	Alive 8 months later	Hochloff	1929	Undetermined origin
19	41	2 children	Amenorrhea; patient received *x*-ray treatment, therefore amenorrhea might be artificial in this case and not due to tumor	Operation	Tumor of the left ovary, metastatic from the left kidney previously removed	Died 5 months after operation	Stadiem	1935	Metastatic

Weiss (1898), Wood 1902, Riche (1914) Fusari (1922) Neuman (1925) Schwoerer (1927) Schiffman, Bovin, Flatau, Weiss, Chairi and Ulrich have reported cases of hypernephromas, majority of which are located in the broad ligament. On some of these cases literature is not available.

and indican gave negative results, no bile was found; trace of albumin was found.

Microscopical examination showed occasional degenerated leucocytes present and many red blood cells. Phenosulphophthalein test: 80 per cent.

After thorough *x*-ray studies in which the spleen was visualized with thorium dioxide and after a bilateral pyelogram, a diagnosis of hypernephroma of the left kidney was made.

On September 30, 1933, the patient was operated through a left lumbar approach, but because of the vascularity and the size of the tumor, it was decided to close the incision and at a future date remove the tumor through a transperitoneal approach. She was discharged October 13, 1933, with the wound well healed.

The patient returned to the hospital November 20, 1933. The tumor had increased in size and was occupying the entire left half of the abdomen. It was firm, irregular, non-tender and only slightly mobile. Another *x*-ray picture of the chest showed no significant increase in the perihilar and peribronchial markings and no evident lesions in the lung parenchyma

November 22, 1933, the tumor was removed through a transperitoneal approach. In the process the external iliac vein was ligated and there was some spillage of tumor material in the wound. She recovered from the operation and was discharged December 7, 1933, as improved.

The patient returned to the tumor clinic at regular intervals and was given deep *x*-ray therapy to the left side covering the anterior, lateral and posterior chest, kidney, pelvic and hip regions. She received some 3,000 roentgen units up to February 5, 1935. She noticed that her menses were irregular, occurring every twenty-one days instead of every twenty-eight days. There was amenorrhea in March and April of 1935, but this may have been due to the *x*-ray therapy. On June 25, 1935, a mass was palpable in the left lower quadrant of the abdomen and she was sent to the hospital.

On admission July 15, 1935, she complained of marked pain in the left lower quadrant of the abdomen and a palpable tumor mass. There had been some loss of weight, but she does not know the exact amount. Otherwise she had been well since her discharge.

Physical examination revealed a rather pale and emaciated person. There were no masses palpable in the region of her previous operation. The examination was essentially negative

except for the pelvis. There was a firm, slightly irregular mass, about 6.5 cm. in diameter, lying posterior and slightly to the left side of the uterus; it was somewhat tender and movable. The uterus was normal in size but pushed forward by the mass. The blood pressure was 134/88, temperature 99°, pulse rate 84 and respiration 20.

Fig. 1. Ovarian hypernephroma bisected.

Clinical laboratory findings at this admission were:

Urinalysis: Acid, specific gravity 1.016, examination for albumin, sugar, indican, acetone and bile gave negative results, no diacetic acid; microscopical examination showed an occasional degenerative leucocyte but no red blood cells were found. The phenosulphthalein test was 55 per cent, but at this time patient had one kidney.

The basal metabolic rate was plus 40 per cent.

Blood Chemistry: Urea nitrogen 12.0 mg. and blood sugar 97 mg. per 100 c.c. of blood.

The sedimentation time was ten minutes; the Freidman test was negative.

Since there were no evident signs of metastasis in the lungs, bones, skin, etc., and as the patient was a fair operative risk, a laparotomy was performed on July 19, 1935, and a large solid tumor of the left ovary was found (Fig. 1). It was not adherent to the surrounding structures and the left Fallopian tube was elongated and stretched over its superior surface. There was no free fluid in the abdomen and the pelvic lymph nodes were not enlarged. However, the lymph nodes were enlarged in the left upper quadrant, the region of her previous nephrectomy. The right Fallopian tube and ovary were removed as a prophylactic measure. She had a stormy convalescence and was discharged August 16, 1935.

Pathological Report. Specimen consists of a globular mass of tissue measuring 14 by 12 by 11 cm. and is covered by a capsule which appears purplish due to the color of the under-

lying structures. The surface of the mass shows a tube attached to one side. There are two small nodules of tumor tissue which are apparently covered by peritoneum near the fimbriated end of the tube. On section, the mass shows a firm but very friable tissue of a light Venetian red color in its periphery and a less friable central portion which is of a pale, cadmium, yellow color (Fig. 2).

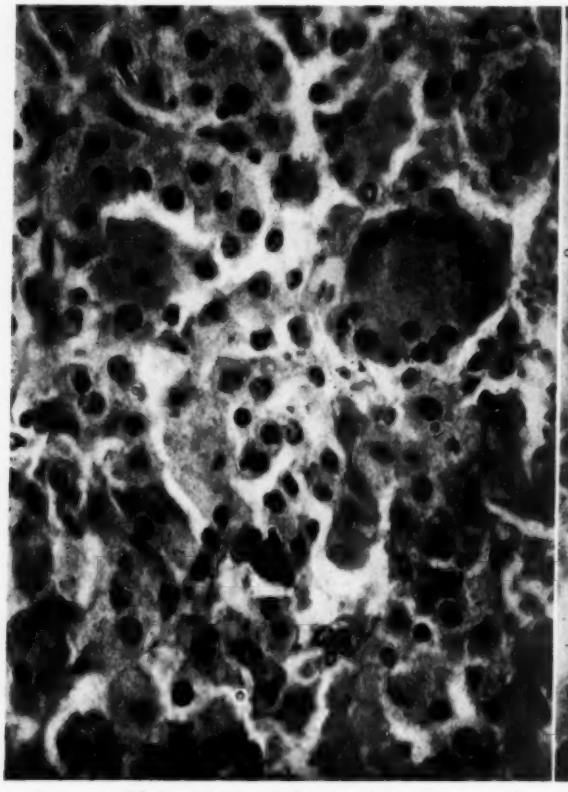

Fig. 2. Photomicrograph showing giant cell.

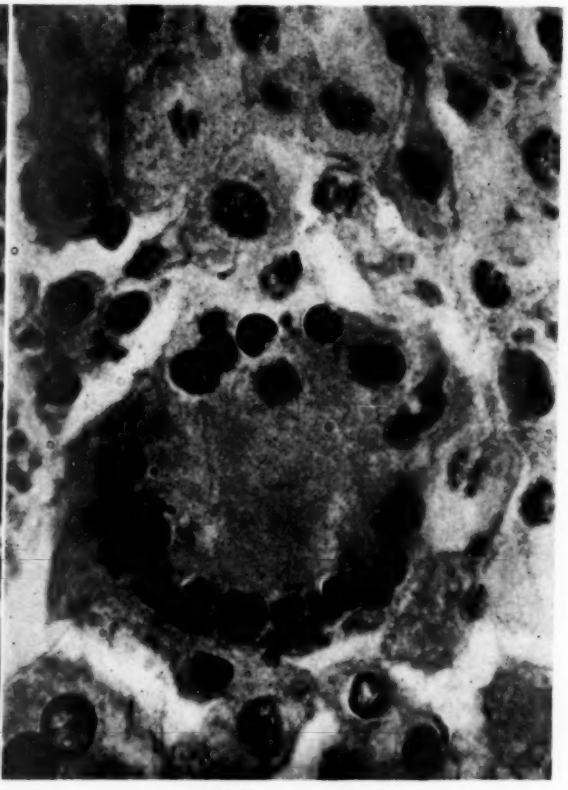

Fig. 3. Higher magnification.

The histology of the neoplastic tissue shows cords of large cells with centrally located vesicular nuclei, some of which have an abundance of pale bluish staining cytoplasm and others have a clear cytoplasm. The former is in the majority. The stroma is very small in amount. Mitotic figures are numerous.

Diagnosis. Hypernephroma of the ovary.

The patient returned to the tumor clinic at regular intervals and in November, 1935, a mass was noted in the liver. She was admitted to the hospital, where she became steadily worse and died December 20, 1935.

DIAGNOSIS

Hypernephroma appearing first as a pelvic tumor is difficult or impossible to diagnose. According to Infantozzi solid tumors of the ovary may be divided into:

A. Connective tissue: fibroma, sarcoma, endothelioma;

B. Epithelia: epithelioma;

C. Heterotopic: embryoma, teratoma, hypernephroma.

In addition to these solid tumors there are some cystic tumors so tense with fluid that they simulate solid growths. There are also the special types of ovarian tumors, such as arrhenoblastoma, granulosal cell carcinoma, dysgerminoma and Brenner tumor. These latter tumors, however, have their peculiar clinical characteristics. The arrhenoblastoma is a masculinization tumor. Granulosal cell carcinomas occur usually after the menopause and are attended with uterine bleeding and enlargement of the uterus with hypertrophy of the breasts. The Brenner tumor is rare but more common than hypernephroma of the ovary.

A review of the tabulated cases reveals that after puberty amenorrhea occurred in

six of the nineteen cases, whereas menorrhea occurred only twice; the menstrual history was not recorded in some cases. When the tumor appeared before puberty, as was the case in three reported instances, there was a premature sexual development with precocity, or virilism. Probably this phenomenon is due to the type of tumor and not to the secondary effects on the ovary. Further substantiation is furnished by Glynn, who noted a precocious growth of body and sex organs, overgrowth of hair and fat, pigmentation rather inconstantly and frequently mental dullness in cases of pure suprarenal hypernephroma.

The menstrual history in cases of other solid tumors of the ovary, as carcinoma, fibroma, sarcoma, etc., depends upon the destruction of ovarian tissue. There appears to be nothing, unfortunately, in the history except perhaps this tendency to amenorrhea, which will aid in the preoperative diagnosis. Probably another added diagnostic hint is that no ovarian hypernephroma has appeared in a colored patient, but most of the reports, however, have been from European clinics, where the Negro population is small.

TREATMENT

Operation offers the only hope of cure. Preoperative irradiation does not seem indicated for metastasis occurs mainly by the blood streams. At the operating table the tumor is to be differentiated from fibroma or any other solid benign ovarian growth, for in the benign type of tumor simple removal suffices. Being a malignancy the hypernephroma is removed together with all pelvic organs.

Exploration of the abdomen is indicated to ascertain if there are any metastatic nodules in the liver and if the ovarian tumor is metastatic from the adrenals.

Intense deep *x*-ray therapy may salve the conscience after removal of the growth but its efficacy is debatable.

PROGNOSIS

The prognosis is poor, for the tumor being blood borne, has a fondness of metastasis; the liver, bones and lungs are the sites of election. Of the nineteen patients, three succumbed to the operation and seven died within nine months after operation. The longest cure definitely reported is four years. Our case died five months after operation with metastatic nodules in the liver.

CONCLUSIONS

1. Hypernephroma of the ovary is extremely rare and is worthy of report.
2. This is definitely a metastatic growth for the same type of giant cells appear in the original tumor.
3. There are no diagnostic symptoms or signs.
4. Removal of a hypernephroma nineteen months before aided in making a correct preoperative diagnosis.
5. Prognosis is poor.

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From—Garrison's "History of Medicine."

MULTIPLE DUODENAL PERFORATIONS*

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THE majority of perforations of the duodenum are due to single ulcers and to trauma from bullet or stab wounds. The principles involved in the diagnosis and treatment of such cases are fairly well standardized in all large surgical services, and would seem to call for no particular comment. In this article we are reporting two cases, in each of which there appeared two simultaneous perforations of the duodenum. In one case two extraperitoneal ruptures were caused by blunt external violence; in the other spontaneous perforations of two separate duodenal ulcers developed.

MULTIPLE TRAUMATIC SUBCUTANEOUS PERFORATIONS OF THE DUODENUM

The liver, colon, transverse mesocolon, mesenteric root and lower thoracic wall afford protection to the duodenum adequate to prevent rupture by blunt violence except in rare cases. A blow of great force only succeeds in its rupture, especially if the victim has a well developed abdominal musculature, as in male athletes who are subject to severe abdominal injuries, these furnishing many of the cases on record.

Tempsky discussed the etiology and mechanism of duodenal rupture, favored by its close fixation to the spine. He distinguished between rupture due to squeezing and that due to direct laceration, and emphasized the difference between intraperitoneal and extraperitoneal perforation.

Marin classifies injuries of the duodenum:

A. According to their degree, as
1. A simple burst,
2. A complete avulsion,
3. An incomplete rupture;

B. According to frequency, rupture of the third, second and first portions;

C. According to trauma, as
1. Contusion, which is favored by the fixity of the duodenum, its fluid content, and the possible incomplete closure of the pyloric sphincter at the time of injury;
2. Tearing: the middle being fixed and the ends movable, the mucosa having most resistance owing to its folds;
3. Compression, whereby the duodenum is pressed against the spinal column, the muscularis and mucosa rupturing first owing to their richness in vessels and poverty in elastic fibers;
4. Pinching of the duodenum between the costal margin and spine.

The latter two mechanisms are most common.

Statistics vary considerably as to frequency of subcutaneous duodenal rupture following abdominal injury. Tschistosserdoff in 1912 reported 52 cases of subcutaneous rupture in the intestinal tract, only one, or 2 per cent of which was in the duodenum, and this at the duodenojejunal junction. Petry in 1896 estimated that 5 per cent of such injuries are in the

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duodenum. Van Hook and Kanaval quote Chavasse as noting seven duodenal ruptures in 140 cases, or 5 per cent, and Voswinckel as finding 2 in 14 cases, or 15 per cent. In 1907 Hertle's estimate was 10 per cent. Although presumably an unusual traumatic lesion, Furtwangler in 1921 gathered 118 cases. Undoubtedly many cases have been overlooked when death has followed abdominal injury and a thorough autopsy not done.

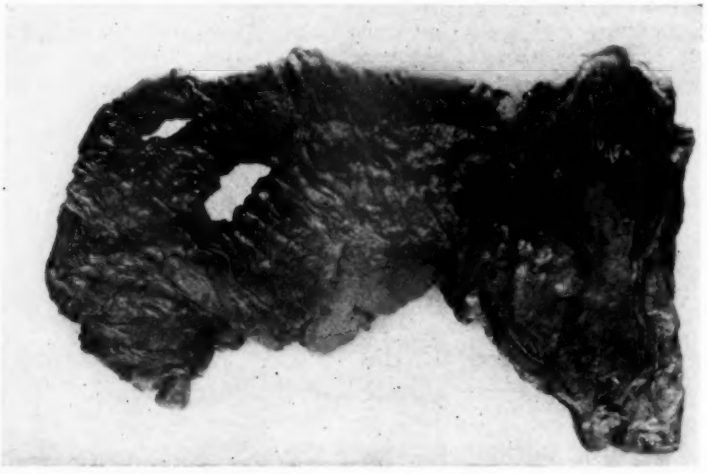

Fig. 1. Duodenum and pyloric portion of the stomach opened longitudinally, showing two duodenal perforations resulting from blunt external violence.

Difficulty in diagnosis has been emphasized by several authors and is illustrated by the case herein reported. Early diagnosis and operation are imperative if the life of the patient is to be saved. Intraperitoneal ruptures produce a primary peritonitis with severe pain, involuntary rigidity of the abdominal muscles, and a silent abdomen, i.e., an absence of the peristaltic sounds on auscultation. Fluoroscopy in the majority of these, as in perforated ulcers, should reveal a bubble of free air or gas between the diaphragm and liver with the patient erect or lying on his left side. This may also be detected by diminution or absence of the normal liver dullness on percussion. On the contrary, extraperitoneal ruptures cause an effusion into the retroperitoneal tissues and may produce tenderness, dullness and even bulging in the right loin, with severe pain extending through to the back, as in Blasker's case. Evidences of peritonitis may be absent in the early stages, developing later due to extension of infection, as discussed by Meerwein; or through physical activity, which increases the size of the perforation or the amount of leakage from it, as in Sereghy's case.

The treatment is surgical, and it is generally agreed that laparotomy is indicated when there is a reasonable suspicion of recent rupture of a hollow intra-abdominal organ. Associated injuries such as laceration of the liver or mesentery must be dealt with as indicated. The important point to remember is the possibility of duodenal rupture and the advisability of an examination of this portion of the intestinal tract, particularly if injuries elsewhere are not found. Any hematoma, localized edema, discoloration, or other evidence of duodenal abnormality should be investigated, as these may be the only intra-abdominal evidences of a retroperitoneal tear. This is well illustrated by Blasker's case, in which a small retroperitoneal hematoma was the only clue to a large extraperitoneal tear in the duodenum. The patient recovered after surgical closure of the perforation and drainage through the loin.

Marin, Amberger and Fischl have also reported cases which have recovered after surgical repair. The cases of Sereghy, Tschistosserdoff and Steinberg died. All have agreed that the mortality is high, being 89 per cent in Furtwangler's report of 104 deaths in 118 cases.

The following case would seem worthy of reporting for several reasons. It is the only rupture of the duodenum caused by blunt violence seen by the authors in a large series of accident cases and in several thousand autopsies where death followed external violence. It illustrates the possibility of multiple (two) retroperitoneal perforations of the duodenum without other intra-abdominal injury, a very unusual finding; and finally, it calls attention to the early diagnostic difficulties in extraperitoneal duodenal rupture and the necessity of considering this possibility in cases of upper abdominal injury.

A colored male, aged 29 years, well developed and nourished, with no significant past history, was injured at work at 4:30 A.M., January 15. He fell about eight feet, landing on his right side and striking the right upper quadrant of the abdominal wall against a blunt object. He immediately experienced severe abdominal pain in this region, and shortly after became nauseated and vomited a brown liquid. He was admitted to the hospital at 5:30 A.M., complaining of moderately severe pain. His pulse rate was 66 and blood pressure 125/82. There was a slight abrasion of the skin of the right upper quadrant of the abdomen, marked abdominal tenderness in this region, and some rigidity. His general condition seemed good and although he vomited several times during the morning he insisted that he felt well enough to go home. At 10 A.M. and again at 4:30 P.M. he was fluoroscoped and carefully examined for the presence of a pneumoperitoneum but no free air was found. Auscultation of his abdomen was not done. Blood count at noon was leukocytes 13,650, erythrocytes 5,580,000 and hemoglobin 85 per cent. The differential count showed polymorphonuclear leucocytes of 84 per cent and lymphocytes 16 per cent. There was definite loin tenderness but no bulging was noted. Urinalysis was entirely negative with a negative benzidine test for blood. Late in the afternoon the maximum tenderness had localized in the right lower quadrant; his pulse rate had risen to 88; the mouth temperature was normal.

An exploratory laparotomy through a low right rectus incision was done under ether anesthesia at 8 P.M. A cloudy blood tinged fluid escaped but no ruptured bowel was found. The operation was hurried because of the patient's condition, and he was returned to his room with a pulse rate of 138, respiration 28, temperature 99°. His condition grew steadily worse and he expired at 9:30 A.M., about thirteen hours after the operation, and twenty-nine hours after injury.

We first saw this case at the autopsy, which revealed hemorrhage and edema in the retroperitoneal tissues about the duodenum and right adrenal gland and in the mesocolon at the hepatic flexure. There was a fibrinopurulent exudate extending up behind the liver and in the subphrenic space, with small pockets of pus in the loose tissues behind the duodenum and around the upper pole of the right kidney. There were no tears in the peritoneum over the duodenum and no definite peritonitis although the abdominal cavity contained some thin bloody fluid. The lungs were quite edematous and there was a seropurulent fluid in the right pleural cavity with a fibrinous exudate on the pleural surface of the right half of the diaphragm. There were no evidences of injury to the diaphragm, thoracic wall, spine, right kidney, pancreas, liver, bile passages, stomach, small intestine or colon. The duodenum is shown in the illustration and a description of it follows:

After opening along the anterior wall the circumference of the pyloric ring was 4 cm. and that of the duodenum 8.5 cm. in its widest portion, which was 9 cm. from the pylorus. Here the duodenal mucosa was brown to red from bruising in an area 6 cm. across and 3 to 4 cm. in the long axis of the intestine. In this area there were two perforations, the larger 2.4 by 1.2 cm., located in the long axis on the posterior wall. Its proximal end was 6.5 cm. from the pylorus. The other, 2.2 by .6 cm., was located more distally in the long axis but on the superior wall. Both perforations had thin, ragged, hemorrhagic, necrotic edges made up of the muscularis. The mucosal edges were also thin and irregular and receded from 1 to 3 mm.

from the openings through the wall. There were no evidences of scarring or chronic ulceration about these perforations or elsewhere in the lining or walls of the specimen. The loose periduodenal tissues were soft, red-brown to black, and partially necrotic. The distal end of the common bile duct was involved in this necrosis and opened into the larger perforation which had destroyed the ampulla of Vater. The loose tissues about the head of the pancreas were soft and discolored but there was no abnormality of this organ itself, and no evidence of pancreatic fat necrosis.

Presumably the perforations were the result of actual compression of the duodenum between the spine and the external object struck in the fall. The findings explained adequately the initial pain and vomiting, the absence of pneumoperitoneum, and the late development of symptoms of infection. About twelve hours after the injury a peritoneal effusion developed, probably from spread of infection into the peritoneal cavity. As this fluid gravitated into the lower abdomen, the maximum tenderness shifted to the right lower quadrant, a rather common occurrence in perforated ulcer, and a reason for this condition being occasionally mistaken for appendicitis. This led to a low right rectus incision, through which the duodenum could not be carefully examined regardless of cognizance of its injury. Death resulted from retroperitoneal infection, right pleuritis and pneumonitis and pulmonary edema.

MULTIPLE SIMULTANEOUS PERFORATED DUODENAL ULCERS

The occurrence of two simultaneous perforated duodenal ulcers is highly unusual. Bager reviewed the records of 1767 cases of perforated duodenal and gastric ulcers and did not mention multiple perforations. None were found by Simon. Multiple simultaneous perforations of gastric ulcers are discussed in detail by Masson and Simon who found records of thirty-two such cases, adding one of their own. They emphasized that the majority, or 72 per cent, of these cases occurred below the age of 30 years, whereas only 53 per cent of single perforated gastric ulcers occur below that age. In their report, abdominal exploration was undertaken in 18 cases, but in only four were both perforations found and closed. The latter four patients recovered. The end result in six others was not recorded. The remaining twenty-three died. These authors conclude that the possibility of a multiplicity of lesions must be borne in mind when dealing with perforated ulcers; and that both walls of the stomach should be examined at the time of operation. Morrison, in 1935, reported 200 cases of perforated gastric ulcer, in one of which there were two perforations.

Recurrent perforations are not at all uncommon in both duodenal and gastric ulcers, and in gastrojejunal ulcers. Cases were described by Henry in 1921 and by Nixon and Lowry in 1928, in which there were five recurrent perforations. Dowdle reported a second acute perforated duodenal ulcer (undiagnosed) in a patient six days subsequent to operation for the first ulcer. Death resulted from peritonitis nine days after the second perforation. Feller discusses the frequency of multiple ulcers and recurrent perforations. He quotes Petreu, Brunner and Brentano in cases of perforation of one ulcer, other or multiple ulcers are present in from 27 to 33 per cent of all cases. Most authors agree that from 5 to 10 per cent of simple uncomplicated duodenal ulcers are multiple. It is, therefore, not surprising that occasionally simultaneous (or almost simultaneous) perforation of two ulcers occurs. Some of the multiple perforated ulcers reported as gastric may have been duodenal though the distinction is of no great importance in either diagnosis or treatment. The average mortality rate in perforated duodenal ulcers is given by Fleming who collected records of 994 patients operated for this condition, with 236 deaths or a mortality rate of 23.6 per cent. He did not mention multiple or recurrent

perforations, but he found only 2 cases in over 1000 in which there was leakage at the site of perforation following surgical closure. Since these operations were performed by various surgeons, his statistics would indicate that the method now in use for the closure of such ulcers is satisfactory. Bager found a mortality of 32.8 per cent in surgically closed perforated gastric and duodenal ulcers. Moynihan in 1919 wrote as follows:

> Not a few cases are recorded in which two perforating ulcers have simultaneously, or almost simultaneously, occurred; or in which, after the secure closure of one ulcer, a second has perforated a week or more later, involving death or a second operation. It is a consideration of cases such as these which has drawn attention to the question of the advisability of gastroenterostomy as an addition to the closure of the rupture in the base of the ulcer.

The mortality in unoperated perforated duodenal ulcers is difficult to estimate with accuracy. In the formes frustes type described by Singer and Vaughan in 1920 this data was not given, but the authors call attention to the probable frequency of the condition, its mild course with usual spontaneous recovery, and also the fact that many perforated duodenal ulcers are undoubtedly overlooked. Usually there is a persistent leakage in acute perforations unless closed surgically, and the mortality must be at least 75 per cent. Kuemmell's mortality on perforated ulcers of twenty-four hours duration was 100 per cent.

Two more dicta may well be added to the list already generally recognized as applicable to the diagnosis and treatment of perforated peptic ulcers, viz.: (1) The advisability of a search for a second perforation at the time of operation for the first ulcer; (2) When symptoms of perforation reappear after surgical closure of a ruptured ulcer, they would indicate the perforation of another ulcer rather than the reopening of the first one. Immediate surgery should be done even if the interval between operations is only a few days.

The following case verifies not only the possibility of recurrent perforation but of simultaneous perforation of two duodenal ulcers. These occurred while the patient was under treatment for a bleeding duodenal ulcer, and illustrate that bleeding ulcers may perforate. Vale and Cameron have recently reported 7 cases of perforated gastric and duodenal lesions, which occurred in the hospital; all were operated and all died. In one of these cases, autopsy showed "perforating ulcers of both the stomach and duodenum." Their experience would indicate that a patient who perforates in the hospital has less chance for recovery than one who is brought to the hospital with perforation.

Mr. T. L., aged 27 years, a clerk, entered the Cook County Hospital complaining of epigastric fullness, pain along the costal margin, and occasional vomiting for about four months. Six years before he had been operated on as an emergency case for a perforation following an illness typical of a bleeding ulcer. He made an uneventful recovery and was free from ulcer symptoms for more than five years. He denied recent hematemesis or tarry stools. He suffered loss of weight and had been on a modified ulcer diet with alkaline powders for about two weeks without relief. Examination revealed nothing of significance except emaciation and an old upper right rectus laparotomy scar. His stools were black and laboratory examinations showed a hemoglobin of 55 per cent and erythrocyte count of 3,090,000.

He was placed on a Sippy diet of hourly feedings of milk and cream and alkalies, and kept in bed. Tarry stools and occasional hematemesis continued but his pain was relieved. Nine days after entrance he complained of "gas pains." His mouth temperature was normal and his pulse rate 90 to 100 during the night. Early the following morning he suddenly complained of severe abdominal pain. Examination disclosed abdominal rigidity, obliteration of liver dullness and absence of peristaltic sounds. Fluoroscopy revealed a moderate amount of free air below the diaphragm on both sides, and he was taken at once to the operating room.

Under ether anesthesia the old right rectus laparotomy scar was removed. There was a diffuse peritonitis, the abdomen containing thin cloudy yellow fluid with fibrin and free air. The duodenum and pyloric area were hidden by adhesions to the omentum and liver. These were separated and the duodenum found to be indurated and covered with a fibrinopurulent exudate. On its anterior wall about 3 cm. from the pylorus was an oval perforation, 5 mm. in length, in the center of a very indurated portion of the intestinal wall. This opening was closed with catgut and linen sutures. It was noted that fluid was still escaping from the superior surface of the duodenum, and when this was exposed, a second, somewhat smaller and slightly more proximal perforation was found. This was also a clean cut opening and its walls only slightly indurated. It was closed by linen and catgut sutures and the duodenum then covered by the greater omentum which was sutured in position. The peritoneal exudate was aspirated and the abdomen closed in the usual way, with drainage to the peritoneum.

There was no postoperative vomiting or distention but his temperature and pulse rate rose steadily and he died of peritonitis about thirty-six hours after operation. Permission for an autopsy could not be obtained.

A careful review of this patient's history suggests that one ulcer perforated when he first complained of "gas pain." It is probable that this opening became sealed off and did not leak extensively, as his pain was only mild until eight hours later when he suddenly developed severe pain and the typical picture of perforated ulcer. This may have signalized the rupture of the other ulcer. Both were found freely open and leaking at the time of operation. The second ulcer might have been overlooked had it not been for leakage of the duodenal contents.

SUMMARY

These two cases illustrate multiple perforations of the duodenum. The first was due to blunt external violence which caused two retroperitoneal tears in the duodenal wall and no intra-abdominal injuries. Therefore, the early picture was not typical of a rupture of a hollow viscus and operation was delayed. Later the peritoneal effusion gravitated to the right lower quadrant with localized tenderness away from the site of injury. The exploratory incision was made too low to visualize the duodenum adequately and the perforations were not found. Death resulted from retroperitoneal and right pleural infection, and the exact pathology was discovered at autopsy.

In the second case the perforations were due to two separate duodenal ulcers which perforated either simultaneously or within a period of eight hours. At operation the second opening was discovered because of the persistent leakage of fluid after closure of the first perforation.

CONCLUSIONS

The possibility of retroperitoneal rupture of the duodenum must be considered after external violence to the upper abdomen. The clinical picture differs from intraperitoneal rupture, and a thorough exploration is indicated. The mortality is very high if the perforations are not closed.

Multiple perforated duodenal and gastric ulcers may occur either simultaneously or nearly so. This possibility should be borne in mind at operation and when symptoms of perforation recur postoperatively.

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The most serious stab wounds of the heart are those which enter the auricles, pulmonary artery, aorta or coronary arteries, as the hemorrhage from these injuries may be rapid. The wounds from the ventricles do not bleed so profusely as a rule, because the contraction of the thick ventricular muscle produces a certain amount of hemostasis.

From—"Legal Medicine and Toxicology" by Thomas A. Gonzales, M.D., and Milton Helpern, M.D.

THROMBOSIS OF THE AXILLARY VEIN

CASE REPORT* WITH COMMENTS ON ETIOLOGY, PATHOLOGY AND DIAGNOSIS

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CASE REPORT

ON July 1, 1936, J. C., male, aged 24 years, came to the clinic complaining of enlargement and hardness of his right upper extremity and prominence of the veins of the right arm as compared with the left. He had been employed for the past year and a half as a clothes presser at which occupation he used his right arm constantly. His past and family history were negative. He was always in robust health and no history of typhoid, malaria, tuberculosis, plumbism, rheumatism, gout, cardiac, or visceral diseases that could affect his condition was obtained.

Three months ago he suffered from a cellulitis of the right leg which was treated by rest in bed and wet dressings for one week. It cleared up without any operative interference. He never was acutely ill after the cellulitis. One month ago, his attention was called to the fact that his right hand was larger than his left and the fingers of his right hand had a darker color than those of the left. The right extremity was never edematous and there was no functional impairment at any time. Several days prior to his coming to the clinic, after studying his reflection in the mirror, he noticed that his entire right upper extremity was larger than the left.

Examination revealed a young, robust, well nourished male, weight 147 lbs., height 65″. General examination was negative save for the right arm. The heart and lungs were normal. Abdomen showed no masses nor enlarged spleen. His reflexes were all normal. The right upper extremity from the shoulder girdle to the fingers was larger than the left but no edema was present (Fig. 1). The color of the skin of the right arm had a slight tinge of rubor. There was a fullness over the anterior chest below the clavicle. The veins over the internal aspect of the arm and in the axilla and the anterior aspect of the chest were more prominent than those on the other side. There was no clubbing of the fingers. No cord was palpable in the axilla and no lymph glands in either the axillary or cervical regions were felt. The veins collapsed slowly on elevation. There was no restriction of motion in the joints. Sensations were unimpaired. Pulsations of the brachial, radial and ulnar arteries were equal in rate and rhythm as compared with the left, with a little greater force in the left. The hand grips were equal. The pulses of the lower extremities were open.

The measurements of both upper extremities revealed the following:

	Right, cms.	Left, cms.
At junction of humerus and shoulder	39.6	33
Midhumeral	30.5	27
Elbow	28.5	26.2
Midforearm	27.5	24.6
Wrist	18.5	16.5
Hand	22.25	20.5

The temperature measured with Electric Potentiometer was equal.

	Right, Deg. C.	Left, Deg. C.
Midhumeral	31.8	31.8
Midforearm	32.6	32.8

The blood pressure on the affected side was less than on the other side.

	Right	Left
Above elbow	90/50	98/60

* From the Peripheral Vascular Disease Clinic of Gouverneur Hospital, New York.

The oscillometric index on the right side was less than on the left.

	Right	Left
Midhumeral	10	11 plus
Midradial	5	6

The venous pressure was markedly increased in the right arm.

	Right, Mm.	Left, Mm.
Cephalic	258	118
Basilic	290	100

The blood count and urine showed no abnormal findings.

The blood Wassermann was negative.

Radiographic examination of the chest, cervical spine and right shoulder failed to reveal any abnormalities.

Venograms of the median cephalic and basilic veins of both right and left upper extremities were taken.

Radiographic examination of the right upper extremity following the injection of 10 c.c. of thorotrast into the median cephalic vein revealed the cephalic vein to be visualized up to the junction of the upper and middle thirds of the humerus at which point numerous venous collaterals were seen on the external aspect of the shoulder region and also supraclavicularly (Fig. 6). The cephalic vein failed to empty directly into the axillary vein. There were also noted two venous segments in the infraclavicular region; the upper one corresponding to the terminal portion of the cephalic vein at the site where it should empty into the axillary vein, and the lower one representing a narrowing of the axillary vein. It received a venous tributary running from the point of the interruption of the cephalic vein across the humerus to be joined by the long thoracic vein. There was also seen another venous collateral coming across just at the level of the head of the humerus and emptying into the same segment.

Examination of the right arm immediately after the first plate was taken revealed that the dye was still present at the junction of the middle and upper thirds of the humerus and several venous collaterals were still visualized

Fig. 1. Photograph of both arms, showing the right larger than the left.

on the external aspect of the shoulder. The two venous segments previously reported were still visualized and the collaterals emptying into it were no longer seen. The veins in the supraclavicular region previously reported and the cephalic vein were no longer visualized (Fig. 7).

Radiographic examination of the left upper extremity following the injection of 10 c.c. of thorotrast into the median cephalic vein, revealed the cephalic vein to empty directly into the axillary vein (Fig. 8).

Examination of the left arm immediately after the previous plate was taken revealed the cephalic vein and the axillary vein to be totally empty.

Radiographic examination of the right upper extremity following the injection of 10 c.c. of diodrast into the median basilic vein showed the basilic vein to be well visualized and distended. The valves were dilated and the vein was tortuous. The axillary vein was not visualized (Fig. 9).

Examination of the right arm immediately after the previous plate was taken revealed the dye to be still faintly present with the valves still visible.

Radiographic examination of the left upper extremity following the injection of 10 c.c. of diodrast into the median basilic vein revealed the basilic vein to be of normal calibre, and the valves smaller in size than on the opposite side.

Examination of the left arm immediately after the previous plate was taken revealed the vessel to be empty save for two spots of dye which were still visualized and corresponded to the site of the valves.

Infra-red photography of the right pectoral and upper arm regions revealed numerous superficial veins (Fig. 10).

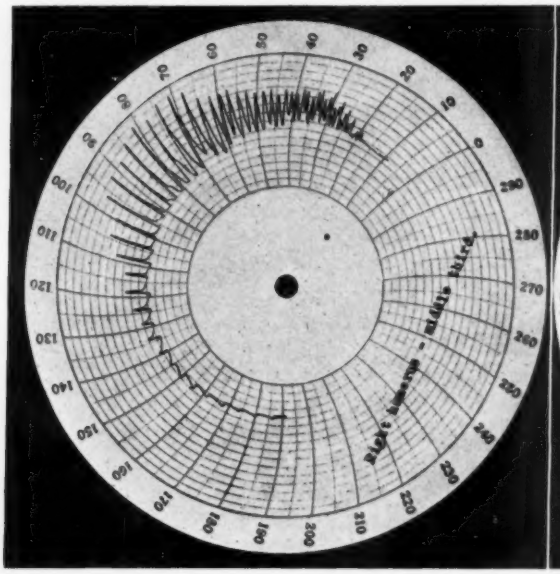

Fig. 2. Oscillometer readings at right mid-humeral region. The oscillometric index is 10.

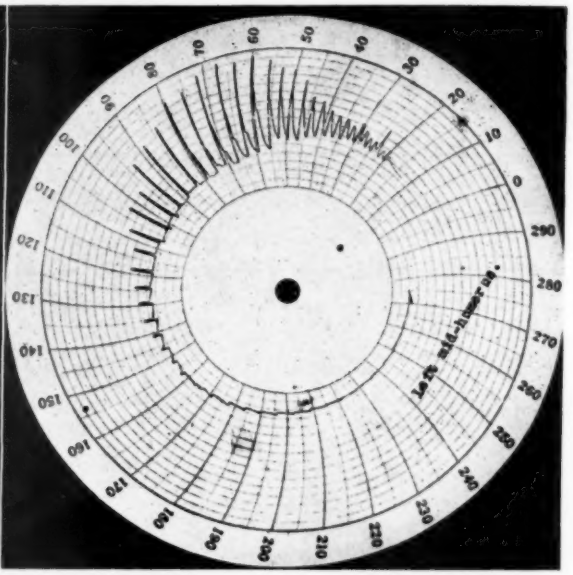

Fig. 3. Oscillometer readings at left mid-humeral region. The oscillometric index is 11 plus.

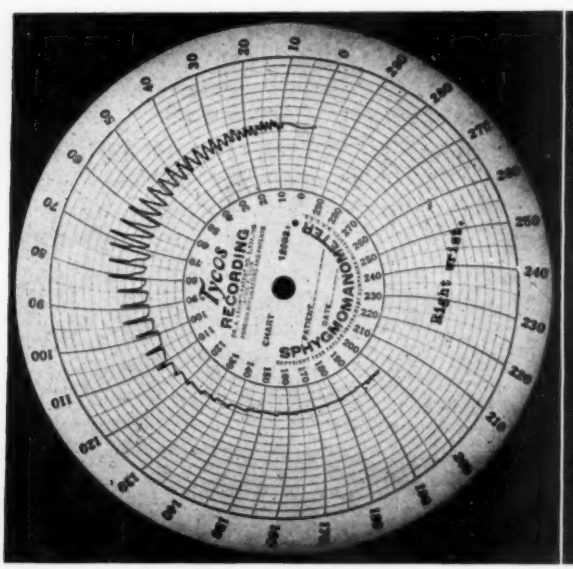

Fig. 4. Oscillometer readings at right wrist. The oscillometric index is 5.

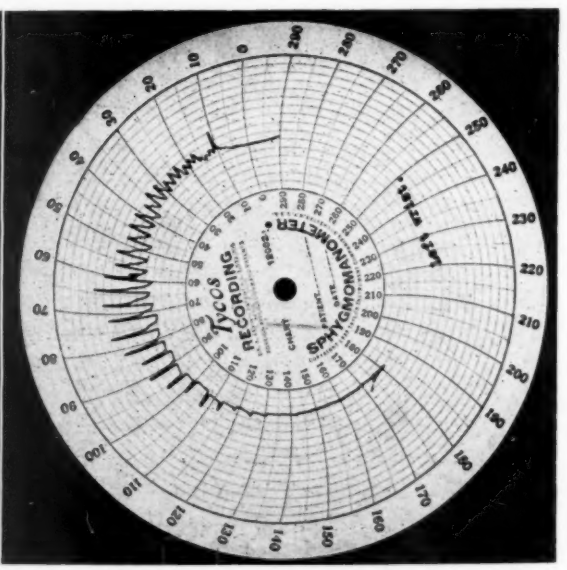

Fig. 5. Oscillometer readings at left wrist. The oscillometric index is 6.

The salient features of this case are:

1. Enlargement of the right upper extremity in a robust, young male, who has worked as a presser for one and one-half years and has used his right arm constantly;

2. Absence of a history of acute pain, associated with a marked edema of right upper extremity resulting in disability;

3. Dilated visible superficial veins of the arm and anterior portion of chest;

4. Cyanotic hue to the skin;

5. Increased venous pressure in both cephalic and basilic veins on affected side;

6. Obstruction to the flow of the dye as evidenced by the failure of the cephalic vein to empty directly into the axillary; the presence of collaterals on the external aspect of the upper arm about the shoulder joint; the presence of veins in the supraclavicular region; the visualization of the long thoracic vein; the distended basilic vein and its valves and delayed emptying as compared with the normal side; and

7. Infra-red photography visualized numerous superficial veins in arm, chest and axilla.

Diagnosis. Incomplete thrombosis of the axillary vein at the cephalo-axillary junction caused by habitual strain.

DISCUSSION

The literature on this subject is rather scant. As a clinical entity, it was first described by Von Schrotter[15] in 1884 who expressed the belief that thrombosis of the vein originated in a sudden stretching and compression after an effort which produced a localized phlebitis with consequent damage of the vessel wall.

Paggi[1] in 1933 reviewed the literature and reported 74 cases. Matas[2] in 1934 reported one case and reviewed the literature, and found approximately 100 cases. Subsequently, about twenty more cases have been recorded by various observers here and abroad.

Reviewing the cases by various authors, one finds that the patients were young, robust, muscular individuals, engaged in heavy work; that males were affected to a greater degree. Females were occasionally affected as in the cases reported by Rosenthal,[3] Cadenot,[4] Finkelstein,[5] Winterstein,[6] Clute[7] and others. The right arm was more often involved than the left and usually those whose left arm was involved either were left handed, or had thrown an unusual strain on the arm. The condition followed an unusual and sudden strain while engaged in heavy work; or developed after severe casual strains such as checking and restraining a wild horse;[8] a fall from a horse on the abducted arm,[9] subsequent to habitual strains as reported by Loewenstein[10] in the case of a waiter who was stricken after waiting on a table, or the woman who was stricken after beating wash clothes with a heavy stick as reported by Rosenthal;[3] resulting from curious causes such as swinging at a golf-ball, pitching a base-ball, or following exercise in a gym as reported by Barker.[11] Sudden and spontaneous development of the thrombosis with accidental strain may develop, such cases having been reported by Pellot,[12] Lohr,[13] Veal and McFetridge.[14]

ANATOMY

The axillary vein is of large size and is the continuation upward of the basilic vein. It commences at the lower border of the tendon of the teres major muscle, increases in size as it ascends by receiving tributaries corresponding to the branches of the axillary artery and terminates immediately beneath the clavicle at the outer border of the first rib, where it becomes the subclavian vein. It is covered in front by the pectoral muscle and costocaroid ligament and lies on the thoracic side of the axillary artery which it partially overlaps. Near the lower margin of the subscapularis, it receives the venae comites of the brachial artery and near its termination, the cephalic vein. This vein is occasionally connected with the external jugular or subclavian veins by a branch which passes from it upward in front of the clavicle.

The vein is provided with a pair of valves opposite the lower border of the subscapularis muscle. Valves are also found at the termination of the cephalic and subscapular veins. Other tributaries are the long thoracic vein, and the costoaxillary veins which come from the first six intercostal spaces, and convey the blood from the intercostal veins to the axillary.

ETIOLOGY AND PATHOLOGY

Predisposing factors such as gout, grippe, rheumatism, lead poisoning, typhoid, and

latent focal infections may be localized in the vein by trauma. As far as syphilis is concerned, the Wassermann in the majority of cases was negative. The exact etiology has not been determined. Several theories as to thrombus formation in the axillary vein have been advanced.

Fig. 6. Radiograph of right upper extremity after injection of 10 c.c. of thorotrast into the right median cephalic vein. At the junction of the upper and middle thirds of the humerus, numerous venous collaterals are seen on the external aspect of the shoulder and supraclavicular regions. Two infraclavicular venous segments are noted. The lower segment receives a venous tributary running from the point of the interruption of the cephalic vein across the humerus, to be joined by the long thoracic vein which is well visualized. There is also seen another venous collateral coming across just at the level of the head of the humerus and emptying into the same segment.

Fig. 7. Radiographs of the right upper extremity taken immediately after Figure 6. The dye is still present at the junction of the middle and upper thirds of the humerus and several venous collaterals are still visualized on the external aspect of the shoulder. The two venous segments are still visualized in the infraclavicular region. The veins in the supraclavicular region are no longer visualized.

1. *Traumatic Theory.* Von Schrotter[15] in 1884 advanced the idea that the thrombosis of the vein was caused by a localized phlebitis which ensued after a sudden stretching and compression of the vein. Loewenstein[10] by means of a series of dissections of thirty-seven cadavers found that with the arm in the abducted position in certain subjects, the costocoracoid ligament together with the subclavius muscle made an indentation into the axillary vein. The axillary vein is distended by abduction or extension of the arm. Venous stasis or circulatory slowing is produced by the forced expiration that accompanies effort. Under these circumstances, pressure on the vein by the costocoracoid ligament furthered by the subclavius muscle is sufficient to effect changes in the vascular endothelium which are in turn sufficient to cause thrombosis of the axillary vein.

Gould and Patey[16] confirmed this observation. They injected plaster-of Paris into the axillary veins of cadavers with the arm in the abducted position. In 2 instances they observed deep grooves from the pressure of the subclavius muscle and in another, a shallow groove corresponding to the position of the costocoracoid ligament. They also noted that a practically constant anatomic finding in the vein at the level of the subclavius muscle was a competent bicuspid valve which was so placed that pressure of the muscle stretched the wall of the vein in the long axis of the valve. Their theory is that the pressure of

the subclavius muscle is responsible for trauma to the vein and that the rupture of the valve is the fundamental lesion, while the position of abduction and the expiratory effort resulting in distention of the vein are predisposing features.

Fig. 8. Radiograph of left upper extremity after the injection of 10 c.c. of thorotrast into the left median cephalic vein. The cephalic vein empties directly into the axillary vein.

Fig. 9. Radiograph of the right upper extremity after the injection of 10 c.c. of diodrast into the right median basilic vein. The basilic vein is distended and tortuous. The valves are markedly dilated.

Veal and McFetridge[14] reported on the basis of roentgenographic and autopsy studies, that the constriction of the vein occurs not as was previously believed over the first rib beneath the subclavius muscle but below the head of the humerus and against the subscapularis muscle. Stretching of the vein takes place within that part of the vein proximal to the point of constriction below the head of the humerus.

Although these facts may be true of cases with marked trauma, it still does not explain the cases where the accidents are too trivial or those rare spontaneous cases without history of injury.

2. *Infection Theory*. According to Matas[2] infection can not be considered as the basis for the thrombosis. He cited 27 cases in which the thrombus was removed and 7 of these were subjected to culture. In 4 cases the cultures were sterile and in the other 3 cases one yielded streptococcus viridans, the second, staphylococcus albus and colon bacillus, and the third, streptococcus. However there were present also changes in the vessel wall indicating a severe inflammatory type of septic phlebitis. The evidence against infection is also the clinical history and the absence of a rise in temperature and other evidences of toxemia.

3. *Sympathetic Theory*. Cottalorda of Marseilles[2] (quoted by Matas) advanced the theory that the lesion originates in a spasm of the veins induced by irritation of the sympathetic venous plexus as a result of trauma. Mere obstruction of the lumen is not the only cause of edema but the persistant irritation of an inflamed or injured perivenous plexus, in the venous wall, is responsible for the rapid edematous swelling.

Although the factors predisposing to a thrombosis are retardation of the blood flow with stagnation, changes in the blood constituents and in the vessel wall, the

exclusive thrombotic theory cannot be maintained in axillary thrombosis since in many instances, no thrombus or actual

Fig. 10. Infra-red photograph of right upper arm, axilla and right chest, showing numerous superficial collateral veins.

occlusion of the vein was found. In a case operated by Cottolorda, no thrombus was found in the vein, but the vein itself was bound in a rigid spasm, obstructing the circulation. However, Paggi[1] reported twenty-eight cases in which an axillary thrombus was found. In some of the cases, the thrombus was found in the subclavian and in others the clot extended from the axillary into the subclavian or from the axillary into the brachial and cephalic and basilic tributaries.

DIAGNOSIS

The diagnosis is based on the following factors.

1. There is a history of an accident or injury by strain or occasionally it may develop spontaneously.

2. There is swelling of the arm immediately or several hours or days after the sudden muscle strain or repeated muscular effort involving the arm. The swelling spreads over the entire arm without any rise in body or local temperature with absence of local inflammatory symptoms and absence of a constitutional reaction.

3. There is a cyanotic hue to the skin.

4. There is development of venous collaterals on the affected arm and over the anterior part of the chest.

5. A tender cord is present in the axilla, though this is not a constant finding.

6. Venous pressure on the affected arm is increased.

7. The oxygen content on the affected arm is lowered.[17]

8. The rate of circulation from the basilic vein on the affected side is increased as compared with the normal side.[11]

9. The blood pressure and oscillometer readings may vary slightly by either showing a slight increase or diminution.

10. Visualization of the veins with opaque medium reveals the presence of numerous collaterals, distended venous valves, and stasis of the dye in the vessels.

11. Infra-red photography visualizes numerous superficial veins in the affected arm, axilla and over the chest.

PROGNOSIS

The prognosis as to life is good. The duration of the disability is variable, as patients may be completely restored in one to two months. In many cases, the convalescence is prolonged because of edema, weakness and stiffness of the arm. Recurrences are noted following exertion.

TREATMENT

The usual treatment of rest, elevation, immobilization with application of pressure bandages, and physiotherapy will clear up this condition. In those instances where the ordinary methods do not improve the condition, thrombectomy or

excision of the venous segment may be required.

CONCLUSION

1. Axillary thrombosis is a definite clinical entity.

2. The etiology has not been definitely proved.

3. Indirect trauma of the axillary vein and of the surrounding structure plays the leading role.

4. Thrombosis of the vessel need not be a constant finding.

5. Physical findings are out of proportion to the extent of the trauma.

6. In cases that resist the ordinary conservative measures, a thrombectomy or excision of the venous segment is indicated.

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BILIARY PERITONITIS WITHOUT PERFORATION*

REPORT OF A CASE AND A REVIEW OF THE SUBJECT

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A WELL developed, well nourished white female of 52 years was admitted to the hospital five days after the onset of a severe attack of pain in the right upper quadrant. The pain was intense, radiated to the back and right shoulder and was accompanied by frequent vomiting. Two days before admission she had several chills and the pain became generalized but remained most marked over the gall-bladder region. There had been no other similar attack.

The patient appeared acutely ill, slightly icteric, the index being plus 25, with temperature of 101.6°, pulse rate 100 and respirations 26. The abdomen was moderately distended, not rigid, generally tender and excruciatingly so over the right upper quadrant. The leucocyte count was 23,100 with 90 per cent polymorphonuclear cells, of which 28 per cent were immature forms.

A provisional diagnosis of acute cholecystitis with perforation was made.

At operation a large amount of thin green fluid, which had the appearance and consistency of bile, welled out of the peritoneal cavity. The omentum and intestines were studded with plaques of various sizes of greenish fibrin. The gall-bladder contained several stones but was neither tense nor soft. It was not covered with gross fibrin nor were there adhesions to it. The walls were thickened and toughened but not edematous. No perforation could be found after a thorough and prolonged examination of the gall-bladder, common duct, stomach, duodenum and jejunum. The pancreas was not thickened or indurated and the presence of such a large quantity of bile in the abdomen could not be explained.

A cholecystostomy was done, all the stones removed and the fundus amputated for pathological examination. Dr. F. D. Bullock reported the microscopical findings as follows:

"Microscopical examination shows a gall-bladder, the wall of which is considerably thickened, the thickening being largely due to an overgrowth of fibrous tissue, particularly marked in the serous and subserous coats, and to extreme hypertrophy of the muscularis. The mucous membrane is atrophic and in many places denuded of epithelial covering; in several areas it is necrotic and hemorrhagic and in a few small regions it is ulcerated, the bases of the ulcers consisting of granulation tissue. This coat shows a number of injected blood vessels and the stroma is moderately infiltrated with inflammatory cells, mostly small round cells. Similar cells are found in the other coats, being in areas fairly numerous. The surface epithelium in places shows proliferative activity with the formation of gland-like or cystic spaces, some of which dip down deep into the wall, several of them extending into the thickened subserous coat. Two of the cysts in this region show partial denudation or desquamation of the lining epithelium and the tissues surrounding them are rather richly infiltrated with inflammatory cells, including a moderate number of polymorphonuclear leucocytes. There is generalized congestion and considerable edema of the outer coats which contain a quantity of fat tissue. Polymorphonuclear leucocytes are fairly numerous in these tunics which also show many small hemorrhages. The outer surface is partly covered with a thin layer of fibrin, in the meshes of which are a number of polymorphonuclear leucocytes."

The patient died on the eighth postoperative day with a terminal bronchopneumonia. Permission for an autopsy could not be obtained.

Prior to 1910, several cases of biliary peritonitis of obscure origin and etiology were reported, the first of these in 1783 by Saunders, and others by Fryer in 1813,

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Barlow in 1844, Bargellini in 1897 and Richardson in 1905.

It was not until 1910, however, that biliary peritonitis without perforation was reported as a possible definite entity by Clairmont and Von Haberer. They first suspected its existence while investigating the effect of common duct ligation upon the renal function in dogs. This procedure was invariably followed by the presence of bile in the peritoneal cavity. Soon thereafter these observations in the dog were substantiated clinically in man. They reported a case where at operation a large amount of bile was found in the peritoneal cavity with no evident perforation of the liver, biliary ducts or intestinal tract. These findings were confirmed at autopsy both macroscopically and microscopically.

They postulated that the bile had filtered through the gall-bladder and bile ducts due to some undemonstrable pathological change in their walls.

Since then there have been some forty similar cases reported in the literature, all foreign except two, and considerable experimental work has been done in an attempt to account for the presence of this bile stained fluid in the abdomen.

Blad ligated the common duct of dogs and injected pancreatic juice into the gall-bladder and thereby produced a complete digestion necrosis leaving it with a well preserved wall structure without inflammatory necrosis.

Schoenbauer and Westphal substantiated these findings and produced gangrene of the gall bladder by the action of trypsin; under these conditions its walls became porous and sponge-like and allowed the bile to permeate through.

Bundschuh reports a case where he found a dilated common duct from stone obstruction at the papilla and an edematous gall-bladder, the surface of which was covered with dew-like drops of biliary fluid which when wiped away, would reappear immediately. The gall-bladder was not perforated and microscopically showed no evidence of inflammation but complete digestion necrosis without nuclear staining. This permitted the biliary contents to filter through. Schivelbein made the same observation in a gall-bladder occluded at the cystic duct.

This explanation of Blad's can only apply to those cases where there is an occlusion of the common duct below the orifice of the pancreatic duct, allowing the pancreatic juices to mix with the stagnant bile in the choleduct and thus find its way into the gall bladder where it produces the same effect as that found experimentally in dogs.

It is interesting to note in this regard that Ravdin, Morrison and Smyth, are of the opinion that penetration of pancreatic juice to the gall bladder in this manner is very unlikely because the bile pressure is greater than that of the pancreatic juice. This view is contrary to that of Harms, which is based upon his experiments on dogs, where manometric readings indicate a greater pressure in the pancreatic ducts than in the bile ducts, especially after the ingestion of food.

These investigators do not agree with the experiments of Clairmont and Von Haberer and suggest that the fatal peritonitis following the ligation of the common duct is probably due to infection from some error in technique or from escape of bile into the peritoneal cavity in sufficient quantity to produce death.

Their experiments confirmed Horrall and Carlson's findings that infection need not be present for a biliary peritonitis to be fatal and that the toxic factor in bile was the bile salts. This fact led them to conclude that the bile stained fluid found in these cases could not be the result of filtration through the walls of the extra hepatic bile ducts because they contained too low a concentration of bile salts.

They liken the common duct and the gall-bladder to the ureter and the kidney pelvis and see no reason why there should be extravasation of bile from an obstructed biliary system and no extravasation of urine from an obstruction in the ureter.

Blad's theory of filtration, on the other hand, is supported by the clinical and laboratory findings of Seifert and Schoenbauer, who found diastase in the gall-bladder bile, and those of Bundschuh, Ruppaner, Popper, Dziembowksi and Brackertz who found these pancreatic ferments in both the gall bladder and the peritoneal bile. Assuming that this retrojection of bile would be possible it would account but for 9 of the 40 cases, i.e., those with common duct obstruction.

Ingebrigsten, in discussing Blad's paper raised the question as to whether the bile stained fluid in the peritoneal cavity might not be the result of the general icterus. In only 5 of these cases was jaundice present. Experimentally, the bile was found in a few days after ligation of the common duct, whereas general icterus was not present. Furthermore the vast majority of these cases were not jaundiced.

Of the other 31 cases, the gall-bladder was described as tense in 21, the walls edematous in 16, thin in 9, without stones in 8 and 4 as jaundiced.

In the thirty-one distended gall bladders from occlusion at the cystic duct, Blad's theory of gall-bladder wall digestion by the pancreatic ferments is hardly tenable when one considers that the cystic duct is occluded, that the veins from the pancreas empty into the portal and splenic veins, and that the lymphatic stream flows towards the pancreas away from the gall-bladder.

In these cases where the action of the pancreatic juice can hardly be considered a factor the changes in the gall bladder may be simply those resulting from the increased pressure of its contents due to cystic occlusion or to infection within its walls.

In one of this type of cases, Bombi reports that microscopical examination revealed an accumulation of bile pigment within the wall where the bile had passed through. This was interpreted as a microscopical perforation because it was found only where there was necrosis.

In two cases considered as biliary peritonitis without perforation, Sick and Frankel, and Nauwerck and Lubke demonstrated, by means of serial sections, microscopical fissures which they felt was the pathway of the bile to the peritoneal cavity and of which there was no visible evidence at operation or autopsy.

It is difficult to conceive that such small openings could be the avenue of escape of large quantities of bile which in some instances is quite viscid.

Sohn unintentionally demonstrated its possibility very strikingly. While operating for stomach ulcer he identified the common duct by puncturing it with a very fine needle through which bile was aspirated. A droplet of bile leaked from this point and a hot moist laparotomy pad was pushed against it until all oozing stopped. Four days later the patient died of a biliary peritonitis and at autopsy a large amount of bile was found in the peritoneal cavity.

At autopsy, there were neither fibrin nor adhesions evident at the site of the exploratory needle puncture in the duct. The puncture wound could not be seen and was found only upon applying pressure to the duct.

Burchardt after numerous experiments on animals has never been able to demonstrate a true transudation of bile or a biliary peritonitis without perforation and all his experiments showed that wounds of the gall bladder or the ducts heal very quickly without leaving much evidence of the lesion. He is of the opinion that at some time or other there was some minute perforation which has since healed. This view is shared by Wolff, Ritter, Meyer-May, Cope, Buchanan, Bundschuh, Hugel, Sick and Frankel and Nawerck and Lubke.

In the cases of Popper, Dziembowski and Brackertz, areas of fat necrosis were seen in the region of the gall-bladder with considerable induration and enlargement of the pancreas and a definite pancreatitis.

In the six other instances where fat necrosis was present there were slight changes in the pancreas in some and none

in others with no real pancreatitis. This necrosis was ascribed to the action of the pancreatic juice in the escaped bile.

In a review of the forty cases published, one is struck by the multiplicity of variations and findings, both at operation and at autopsy.

The different stages at which these come for surgical attention may account to a certain extent for the great difference in clinical and pathological pictures but give no clue to their causes.

In all of the cases showing fat necrosis the pancreas was definitely examined and no extensive lesion was reported. In twelve, no mention was made of the pancreas and in the others no abnormality was noticed.

CONCLUSIONS

1. Biliary peritonitis without evident perforation cannot be explained on any single basis but probably results from different causes.

2. In obstruction at the papilla of Vater it may be due to pressure in the duct and gall bladder in conjunction with a digestion necrosis from the action of activated pancreatic juice, as brought forth by Blad.

3. In occlusion of the cystic duct, the change produced in the gall-bladder wall by pressure and distention allows the bile to filter through, as demonstrated by Clairmont and Von Haberer.

4. In other instances there is some minute perforation of the biliary tract which is invisible, or has healed over, as shown by Sick and Frankel, and Nauwerck and Lubke.

5. Fat necrosis when present is due to the action of pancreatic ferments in the peritoneal bile or to an associated pancreatitis.

6. In the few jaundiced cases the bile stained fluid could conceivably be part of the general icterus.

7. The diagnosis most frequently made was that of acute cholecystitis or appendicitis with generalized peritonitis.

8. On account of the very poor condition of these patients the treatment consists in the simplest procedure which will drain the biliary tract.

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In a third degree burn that is completely free from infection, the necessity of skin grafting is greatly diminished. This is due to the fact that in the majority of third degree burns, even though the full thickness of skin is destroyed, there are thousands of little islands of epithelium at the base of the sweat glands and hair follicles that will grow and spread if they are not bathed in pus.

From—"Preoperative and Postoperative Treatment" by Robert L. Mason, M.D.

NEW INSTRUMENTS

A DEVICE TO FACILITATE SURGICAL DRESSINGS*

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THE management of surgical wounds obviously entails surgical procedures suited to each individual case. Assuming that the surgeon has properly treated the lesion, the seemingly unimportant problem of surgical dressings arises, which, because it appears to be of secondary importance, has received little of the attention it urgently requires.

Only too frequently wounds are dressed with a so-called dry dressing or covered with some inert, though harmless, unguent. In some cases the course and length of convalescence may not have been materially affected but in handling a large series of wounds of many types and of varying degrees of infection, such haphazard after care will result in an appreciable increase of unnecessary hospital or clinic days; to say nothing of the attending loss of working days or of efficiency in the worker.

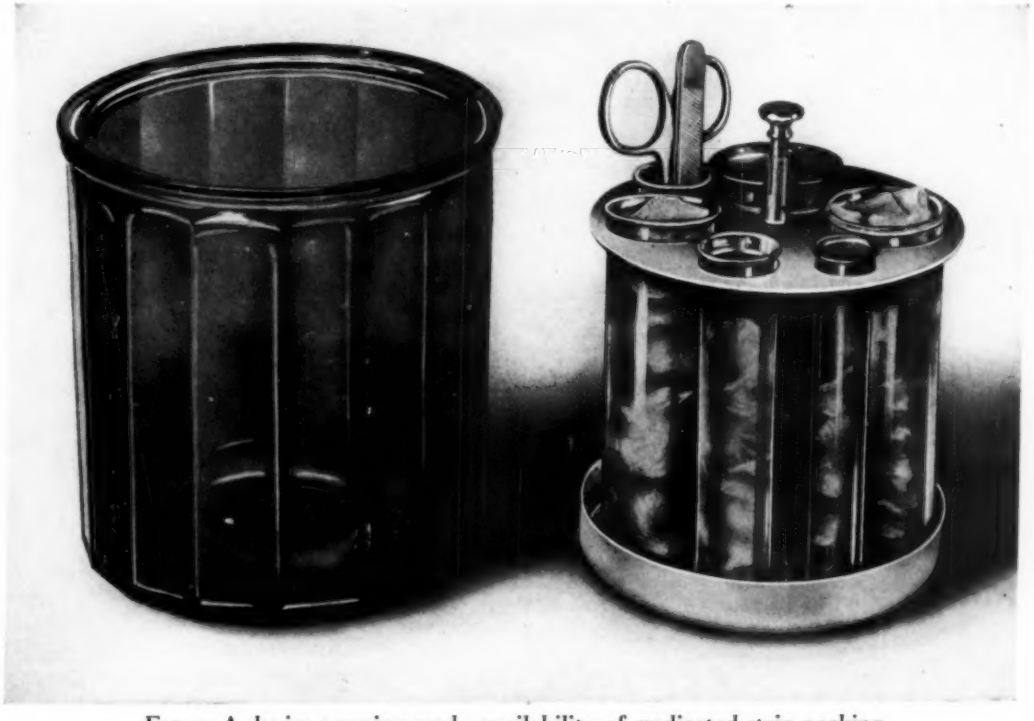

FIG. 1. A device assuring ready availability of medicated strip packing.

In a busy surgical out-patient department or industrial surgical dressing room, it has been found that the use of a simple

* From the Fourth Surgical Service, Bellevue Hospital, Dr. Carl G. Burdick, Director.

device shown in Figure 1 will permit handling of cases with a great saving of time and effort.

The device is enclosed in an amber colored jar with a removable cover. It consists of a metal stand of duraluminum or plated metal supporting a circular plate in which there are openings of varying sizes to hold lipped amber tubes of convenient diameters. These tubes are filled with gauze packing of varying widths, the sizes most commonly used being ¼ inch, ½ inch, 1 inch and 2 inches. The tubes may be filled in the surgical supply room and autoclaved with other gauze supplies. They are then placed in the metal rack and azochloramid in triacetin 1:500, is slowly added until all the gauze has become thoroughly saturated and a slight excess of the solution remains at the bottom of the tube.

In this way we have available continuous lengths of varied widths of "ready-to-use" medicated dressings. One tube supported in the rack contains 70 per cent alcohol, to hold a scissors and thumb forceps, with which the desired length of gauze packing may be cut.

We would like to comment briefly on the medication, azochloramid. This new antiseptic has received favorable comment in the reports of a number of investigators and our own experience with this stable and non-irritating chlorine bactericide extends over three years. In packing the tubes we employ azochloramid in triacetin 1:500, as this solution is slightly oily (preventing adherence to dressings) and will not dry out. It is so stable that dressings remain effective on the wound surface for twenty-four hours or more, and as the device which we are describing is peculiarly suitable for the out patient department, such a medication is well adapted to use with it.

We have employed a device of this sort for the past year on the Fourth Division Surgical Out-Patient Department of Bellevue Hospital and have found it to be very useful. It is inexpensive and a great time saver. We believe that it might be used advantageously in clinics, surgeries of large industrial plants, and as a part of the equipment of dressing carriages on surgical wards.

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A NEW TYPE BIOPSY NEEDLE*

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BIOPSY, regardless of the method, is fraught with the danger of spreading malignancy. We must, however, admit that diagnosis is of primary importance and in selecting a method, one which is the least attended by the danger of metastasis should be chosen.

To this end a new type of aspiration biopsy needle is now being used successfully at the Tumor Clinic of the Caledonian Hospital. By this method we believe that a maximum amount of tissue is obtained with a minimal of trauma. The tumor is punctured only to a slight extent not necessitating the usual "Y" puncturing, and the needle operates on hard and soft tumors equally well.

The needle consists of an outer standard gauge needle, with an inner needle to fit the bore of the outer needle closely. They are 3 inches long, and when one is placed into the other the points of both form a smooth, even bevel which for the purpose of description we will call position 1. In this position the hilt of the inner needle has a single peg which fits into the notch on the hilt of the outer needle.

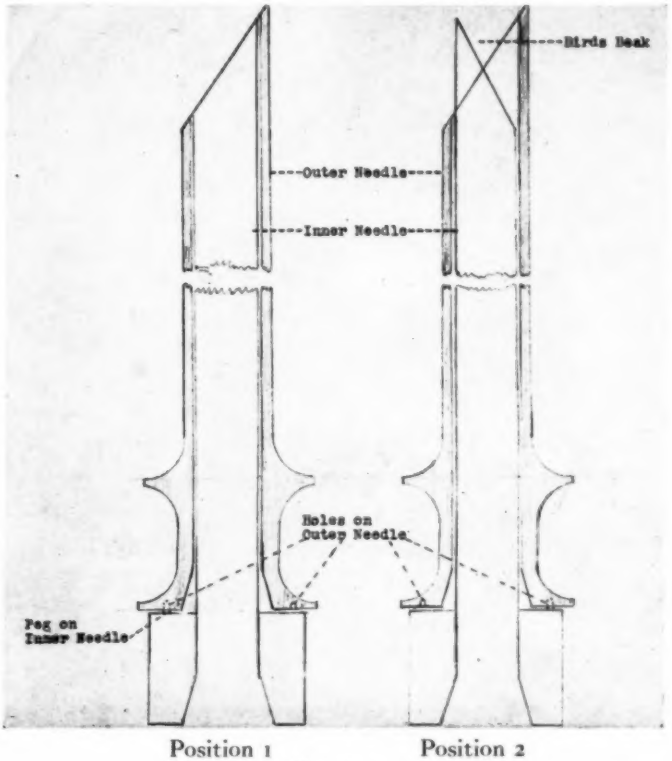

FIG. 1.

It is apparent that by withdrawing the inner needle enough to clear the notch on the outer needle, one can be rotated in the other. Our needle is so modified that we can accomplish this rotation through 180 degrees, so that the peg on the inner needle will come to fit into another notch on the

* Presented at the meeting of the Local Chapter of American College of Surgeons in King's County Hospital on November 14, 1936.

hilt of the outer needle and hold them in this position. This second position, for ease of description, we will call position 2. In this latter position the bevels of both needles are opposite each other, forming a sort of bird's beak.

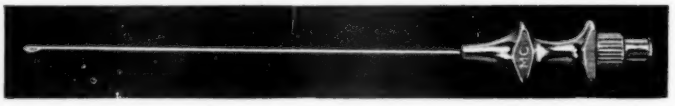

Fig. 2.

The needle is used in the following manner. The skin over the tumor is sterilized, and the needle with syringe attached is inserted with its component parts in position 1. When the tumor mass proper has been penetrated, the inner needle and syringe is slightly withdrawn and rotated through 180 degrees in either direction. This puts the needle in position 2. Now both needles in this position are pushed into the tumor and at the same time giving them a rotary movement. By doing this the beak of the needle has traveled in a straight and rotating path. By using suction on the syringe at various phases of the rotary course of the needles, more than ample material is obtained for microscopic examination.

It is a good plan to withdraw the inner needle first, at the same time using suction so as to pick up as much of loosened tumor cells as possible, and then do the same with the outer needle as it is withdrawn, thereby attempting to lessen as much as possible the chance of cell extension and metastasis.

CESAREAN SECTION: INSTRUMENTS FOR LOW CERVICAL OPERATION*

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THE performance of the low cervical cesarean operation is facilitated by the use of the two instruments herein described and illustrated.

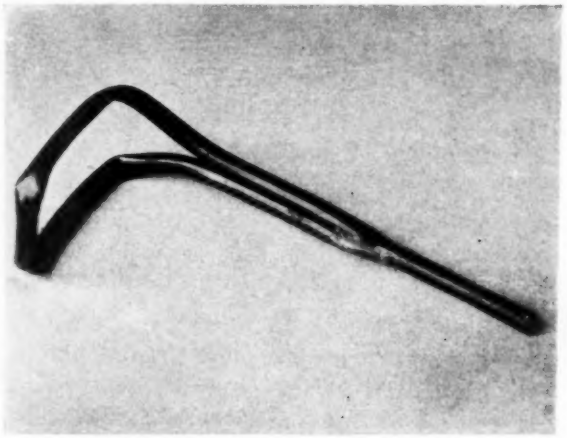

FIG. 1. Combination bladder retractor and amniotic fluid aspirator.

COMBINATION BLADDER RETRACTOR AND AMNIOTIC FLUID ASPIRATOR

This consists of a tubular metal retractor with a series of rows of holes extending from the surface to the cavity at the distal edge of the blade (Fig. 1). The rubber tubing of the aspirating pump is attached to the end of the handle so that when the bladder is retracted and the incision in the lower uterine segment made the amniotic fluid and the spilled blood are automatically sucked up through the handle. The blade of the retractor, being at the lowest portion of the incision, keeps the field of operation exceptionally clean without additional aspiration.

FIG. 2. Vectis for extraction of the fetal head.

VECTIS FOR EXTRACTION OF FETAL HEAD

This is a flat strip of rigid metal 1½ inches wide, shaped somewhat like a Dever retractor, but the handle is bent farther back (Fig. 2). With the incision in the lower uterine segment made, a finger of the left hand is inserted into the infant's mouth and its face pulled up to the opening. The curved blade of the vectis is then inserted into the uterine cavity around the head which is gently lifted out. This obviates the use of forceps or undue stretching of the incision by a hand under the head.

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SURGICAL PROBLEMS

This department, for which the answers have been prepared by competent authorities, is maintained for the benefit of our subscribers. All queries must contain the name and address of the writer. Names will not be published.

Question: A patient's urine contains shreds, especially the first specimen in the morning. He has no urethral discharge. About ten years ago he had a Neisserian infection which was mild in type. What should I do for this man? Please omit name.

Answer: These shreds are usually the remnants of a former infection, either from the prostate, the lubricating glands of the urethra, or granulation tissue from an erosion usually back of a stricture band. Have the patient urinate and gently irrigate his anterior urethra with 1:2000 Silvogon. Then make a smear of the prostatic secretion. Following this insert a bulbous bougie, size 24, French, to ascertain if there are any stricture bands present in the urethra. Should the prostatic secretion be free of pus the treatment will be the passage of curved sounds beginning at the calibre of the stricture and gradually increasing until size 28, French, is reached. Follow by irrigations of 1:4000 silver nitrate weekly. Should pus exist in the prostatic secretion, the treatment should alternate with bladder instillations of 1:4000 silver nitrate followed by massage of the prostate. It is not advisable to massage the prostate and pass curved sounds at the same sitting.

Question: Stone has passed from the kidney down to lower portion of the ureter and has been lodged at the present site for the past three months. Patient continues to have colics which require morphine sulphate but stone does not move into bladder. Please outline treatment.

Answer: This type of case is very often aided by incising the mouth of the ureteral meatus through a cystoscope and the passage of a ureteral catheter (two, if possible), so as to stretch the ureter and possibly change the axis of the stone, that it may descend more easily. If catheters are able to be passed beyond the stones it is advisable to allow them to remain in place twenty-four hours, and before removing inject 2 c.c. of mineral oil in order to facilitate passage of the calculus. High vitamin A diet, easily secured by the addition of cod liver oil to the ordinary diet, is advisable for the patient with urinary stones.

Question: I have a case of long-standing pyuria which did not clear up under ordinary urinary sedatives and antiseptics. *X*-rays were taken which showed large stone in both kidneys. What should I advise the patient to do?

Answer: This type of case is precarious. I feel that an intravenous urography should be performed, which by the amount and density of the dye will indicate the best-functioning kidney, and how much structural damage has been done by reason of these stones. I would suggest that the best side be operated upon for calculi removal, and that a period of three months interval should be allowed before operating upon the other side, in order to allow the kidney just operated upon to reach its maximum efficiency. High vitamin A diet and general measures are necessary, and if marked anemia is present transfusions are indicated.

Question: A man has an ovoid lump which is smooth and freely movable in the region of the right external ring. There is no impulse on coughing, nor can I reduce it. It is well above the testicle. The patient states that the size varies from time to time. Could you offer any suggestions as to the handling of this case?

Answer: The most logical deduction from your description is that of a spermatocele.

Place the patient in a dark room, and with a small flashlight transilluminate this mass. If the light is transmitted easily through it, the diagnosis will be assured. It is also possible that it might be a cyst of the globus major of the epididymis, which would also transmit the light. Treatment is aspiration of the fluid through a 20 gauge intravenous needle under asceptic precautions. Be sure that all the fluid has been withdrawn, then instill 2 c.c. of 5 per cent quinine and urea hydrochloride and withdraw the needle. One injection is usually sufficient, but occasionally we have to repeat this process a second or third time.

Question: How should the following case be managed? Primipara, age 22, pregnant at term and beginning labor, the membranes having ruptured two hours previously. Pelvic measurements were normal, head "dipping" in pelvis, cervix 2 cm. dilated, baby apparently normal size or smaller, position not made out, fetal heart sounds were excellent.

This woman had had a diagnosis of moderately advanced pulmonary tuberculosis when she was three months pregnant, and interruption had been advised. She disappeared and was not seen again until labor began as noted above.

Answer: The combination of pulmonary tuberculosis and pregnancy calls for expert advice and care, preferably by a tuberculosis specialist and an obstetrician interested in tuberculosis working in close coöperation.

The case cited was evidently one of active pulmonary tuberculosis at the beginning of pregnancy, and therefore should not have continued with the pregnancy. Evidently this advice was given but refused by the patient or her family. Consequently, the obstetrician was obliged to handle the case as best he could when called in after the membranes had ruptured and labor was in progress.

There were two courses open to the obstetrician in this case, viz: (1). Allow labor to continue and when full dilatation of the cervix was accomplished, application of forceps and immediate delivery, preceded by episiotomy. (2). If for pelvic reasons—e.g. contracted pelvis, large baby etc.—spontaneous delivery seemed improbable, Cesarian section should have been performed. Since there was apparently no disproportion in this case, delivery from below should have been the method of choice. Local anesthesia in either of these methods is desirable.

Naturally, labor in such cases should be made as painless as possible by the use of some form of obstetrical analgesia. We use nembutal (pentobarbital), and morphine or scopolomine. As soon as the cervix is out of the way, forceps should be applied and delivery accomplished without delay. The longer the patient is in labor the worse for the pulmonary infection. If for any reason local infiltration or "block" anesthesia cannot be used, nitrous oxide and oxygen, or clyclopropane is indicated. Never use ether or chloroform.

The baby, of course, should not nurse in such a case. The mother, after delivery, should be treated as any non-pregnant case of active pulmonary tuberculosis, which means proper "curing until arrest" has taken place.

Question: Am considering getting my radium from ——— Company. On the twenty-four hour service they state that they can supply only Radon, and indicate that 100 millicurie dose for twenty-four hours will be the same as 100 milligrams for twenty-four hours. Do you advise this substitution? I want to use it either intra-uterine after diagnostic D & C, or in Clark tube for cross fire; dosage to be as indicated.

Answer: The main difference between radium element and radon is that about half of radon is used up in four days, whereas the activity of radium element persists indefinitely. As about 15 per cent of radon is lost in twenty-four hours, an initial radon content of 100 millicuries active for twenty-four hours is less than an initial content of 100 milligrams of radium element, active for twenty-four hours. I feel that it is wrong to give long distance advice as to methods of radium therapy and therefore do not wish to tell anyone what to use. Radium treatment is influenced by so many factors that long distance advice is not only useless but dangerous.

BOOK REVIEWS

UROLOGY FOR NURSES. By Oswald Swinney Lowsley, M.D., F.A.C.S. and Thomas Joseph Kirwin, M.D., F.A.C.S. J. B. Lippincott Company, Philadelphia and London: 1937.

This is a practical book designed to outline the subject with the nursing factors presented in detail thereby making it unnecessary to constantly refer to books written for physicians and medical students.

It covers the following subjects: Historical Review, The Social Psychology of the Nurse, Technical Procedures, Urological Instruments and their Care, Cystoscopy and Radiography, The Kidney, The Ureter, The Bladder, The Prostate, The Male Urethra, Cowper's Glands, The Seminal Vesicles and Vasa Deferentia, The Scrotum and Its Contents, The Penis, Urology in the Female, Diet, and a Glossary of Urological Terms.

In its 520 pages there are 101 illustrations and an Index.

A good book for nurses, and may we add, also for medical students.

PHYSICAL THERAPEUTIC METHODS IN OTOLARYNGOLOGY. By Abraham R. Hollender, M.D., F.A.C.S. With 189 illustrations. St. Louis: The C. V. Mosby Co., 1937. Price $5.

Eleven authors, all well known in their field, have contributed to this book of 442 pages.

The author felt that many otolaryngologists, although interested in physical medicine, have in part failed to avail themselves in its benefits, probably due to a lack of familiarity with the underlying principles and techniques. This volume is an earnest and successful effort to fill such a requirement. A small part of the text is devoted to fundamentals, but the main body of the book considers the clinical problems encountered in everyday practice.

Part One deals with Physical Agents, Principles, Effects, and discusses such topics as Low Voltage Currents, Diathermy, Short-wave Diathermy, Electrosurgery, Photothermy, Ultraviolet Irradiation, and *X*-rays and Radium.

Part Two has chapters on Affections of the External Nose and Face, Acute Rhinitis—Acute Sinusitis, Ionization in Simple Chronic Rhinitis, (also, in Vasomotor Rhinitis), Diathermy in Reduction of the Inferior Turbinates, Chronic Sinusitis, Roentgen and Radium Therapy in Hypertrophied Tonsils, Electrosurgical Tonsillectomy, Laryngeal Tuberculosis, Affections of the External Ear, and Hearing Aids.

In Part Three the authors deal with Endoscopic Approach to Therapy, Benign Neoplasms, Malignant Neoplasms and Miscellaneous Conditions, such as Epistaxis, Facial Paralysis, and so forth.

We have not given every chapter heading, but feel we have listed enough for the reader to get a comprehensive idea of what the book covers.

An excellent, well written work.

A MANUAL OF RADIOLOGICAL DIAGNOSIS FOR STUDENTS AND GENERAL PRACTITIONERS. By Ivan C. C. Tchaperoff, M.A., M.D., D.M.R.E. (CAMB.), Assistant Radiologist and Radium Registrar, St. Thomas's Hospital, London. With a Foreword by Philip H. Mitchiner, M.D., M.S., F.R.C.E., Surgeon to St. Thomas's Hospital. Pp. 255, with 285 illustrations. Baltimore: William Wood & Company, 1937. Price $6.00.

In this work the author has attempted to present a synopsis of the essentials of radiological interpretation, intended for the student in curriculum as well as for the special student and the general practitioner who is expected to understand the reasoning on which the expert radiologist bases his report and to coordinate it with his clinical findings. Emphasis is laid on the necessity of following a systematic scheme in the analysis of the films with careful attention to details. In addition to the commoner pathological states, the author includes most of the rareties.

The illustrations deserve special mention because of their generous size and instructive captions. They are printed in the negative

which is perhaps better in a textbook. Most of the illustrations are patently representative of the conditions they are captioned to exhibit; in a few instances, the reviewer would have been more content had the author mentioned how the final diagnosis was reached.

The text deserves special mention because of its conciseness. The tabular form so often followed in the work lends itself admirably to teaching purposes. This would really be an excellent textbook for the teaching of radiological diagnosis.

Clinical Roentgenology of the Cardiovascular System. By Hugo Roesler, M.D. Springfield, Ill.: Charles C. Thomas, 1937. Price $7.50.

The author in his Preface reminds us that an "examination of the cardiovascular system by means of the roentgen rays is but one among several methods of study." He further states that, "The examination [of the heart] by roentgen rays has rightly been called an autopsy performed on the living." In his book, Dr. Roesler has added a valuable contribution to medical literature. The beginner may have trouble with parts of the text, but on the whole the man doing work in this field will find the book a practical aid.

In this work of 343 pages the author covers Technique, Anatomy and Roentgenology, Normal Cardiovascular System, Measurement, Dynamics of the Normal Cardiovascular System, Anatomical-Clinical-Roentgenological Considerations, Cardiovascular Disease from the Viewpoint of Etiology (and) from the Viewpoint of Structural Changes, Diseases of the Aorta, Diseases of the Pulmonary and Its Branches, and Pericardial Disease.

There are a Bibliography (at the end of each chapter) and an Index. The work is well illustrated. All in all, a well done, practical, needed work.

Medical Morals and Manners. By Hubert Ashley Royster, M.D. Chapel Hill, N.C.: The University of North Carolina Press, 1937. Price $2.50.

Under the title, Medical Morals and Manners, the opening essay, Dr. Royster has included the papers and addresses which he has written and published during the past forty years, a period devoted by the author to the practice of surgery. Some of them were delivered before lay audiences, a number appeared in newspapers and magazines, some constitute addresses presented before medical societies, a few are entirely non-professional, and none of them is technical.

Dr. Royster has covered a wide variety of subjects, and writes with a facile pen.

Personally, we enjoyed reading this book. We feel that others, also, will find it stimulating reading. A book to have in your library, to pick up at odd moments, and to return to it many times.

Diseases of the Newborn. Abraham Tow, M.D. New York: Oxford Medical Publications, 1937.

This is a well written book which is an attempt to organize the contributions made by embryologists, pathologists, chemists, surgeons, obstetricians, and pediatricians in such fashion that it will be of service to the general practitioner.

In general the Contents cover: General Considerations, Infant Feeding, Premature Infant, Birth Injuries, Skeletal System, Digestive System, Respiratory System, Circulatory System, Blood Dyscrasias, Genitourinary System, Diseases of the Nervous System, Diseases of the Eye, Nose and Ear, Infectious and Septic Diseases, Diseases of the Skin, and Special Topics, such as Thyroid, Mongolian Idiot, Vitamin Deficiency Disorders, and Offspring of Diabetic Mother.

There are 52 illustrations, a Bibliography, and an Index.

The young man early in the practice of medicine, and the general practitioner, will profit immeasurably by studying its pages.

Inhalation Anaesthesia. A Fundamental Guide. By Arthur E. Guedel, M.D. New York: The Macmillan Company, 1937. Price $2.50.

This is one of the Surgical Monographs edited by Elliott C. Cutler, M.D.

The author presents an outline intended to guide the anesthetist up to and through his first few general anesthetics. The writer tells us "it is not complete in detail save where detail is necessary, as in 'Signs of Anaesthesia.'" It intentionally treats lightly and more complicated physiological and pharmacological

mechanisms, describing these only to the extent necessary in their clinical application."

Part One considers the Principles of Inhalation Anesthesia, and Part Two Anesthetic Accidents.

To the physician who must at times give a patient an inhalation anesthesia but who admits (at least, to himself) that often he loses his way, Dr. Guedel has written just the book that man needs. He who reads it, if he applies all that is taught, will be a fairly safe man to administer an anesthetic in an emergy, or when an expert anesthetist is not at hand. A good book for medical students.

THE LUNG. By William Snow Miller, M.D. Springfield, Ill.: Charles C. Thomas, 1937. Price $7.50.

This excellent and valuable monograph is the result of forty-seven years' study of lung structure by the author. He began this labor in 1887 while working in the laboratory of the College of Physicians and Surgeons. At that time Francis Delafield was advancing the thesis that free communications existed between the air spaces in the lungs. In this he was supported by J. West Roosevelt. Dr. Miller was so impressed that he made it the subject of his work at Clark University. Since then, at the University of Wisconsin, and in addition at Johns Hopkins and Leipzig for a year each, he has continued at this work.

The book embraces chapters on The Lungs, The Trachea and Bronchi, Intrapulmonary Bronchi and Bronchioli, The Air Spaces, The Blood Vessels, The Lymphatics, The Pulmonary Lymphoid Tissue, The Nerves, The Pleura, Key Points, Historical Sketch, and The Acinus.

There is a comprehensive Bibliography, and an ample Index. There are 152 illustrations gathered from several sources.

The internist and surgeon might well read this sane and authoritative work.

OPERATIVE SURGERY. By J. Shelton Horsley, M.D. and Isaac A. Bigger, M.D. With contributions by C. C. Coleman, M.D., John S. Horsley, Jr., M.D., Austin L. Dodson, M.D., and Donald M. Faulkner, M.D. in two volumes. Fourth Edition. Illustrated by Miss Helen Lorraine. St. Louis: The C. V. Mosby Company, 1937. Price $15.

This well known Operative Surgery is practically a new work.

Dr. Horsley invited Dr. Bigger, Professor of Surgery at the Medical College of Virginia, to act as co-author of the book, and he is responsible for the chapters on surgery of the neck, thorax, breast, hernia, nervous system, and some of the operations upon the extremities.

Dr. C. C. Coleman, Professor of Neurological Surgery at the Medical College of Virginia, wrote the surgery of the central nervous system and the cranial nerves. Dr. A. I. Dodson, Professor of Urology at the Medical College of Virginia wrote on urology, and Dr. John S. Horsley, Jr., did the parts on plastic surgery. Dr. Donald M. Faulkner covered orthopedic surgery. Dr. Guy W. Horsley gave much aid in the preparation of the chapter on proctology.

Many new operative procedures are described which have not heretofore been published in a book.

More than 500 new illustrations have been added to this edition by Miss Helen Lorraine.

References are at the conclusion of each chapter. The two volumes are 1387 pages long. There is a good Index.

This work deserves to be well received by the profession.

PRACTICAL CLINICAL GYNECOLOGY

HENRY C. FALK, M.D., F.A.C.S.

SIXTH INSTALLMENT

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Chapter IX

OVARY

In order to have a clear comprehension of the normal and pathological changes in the ovary, a knowledge of its embryology and physiology is essential.

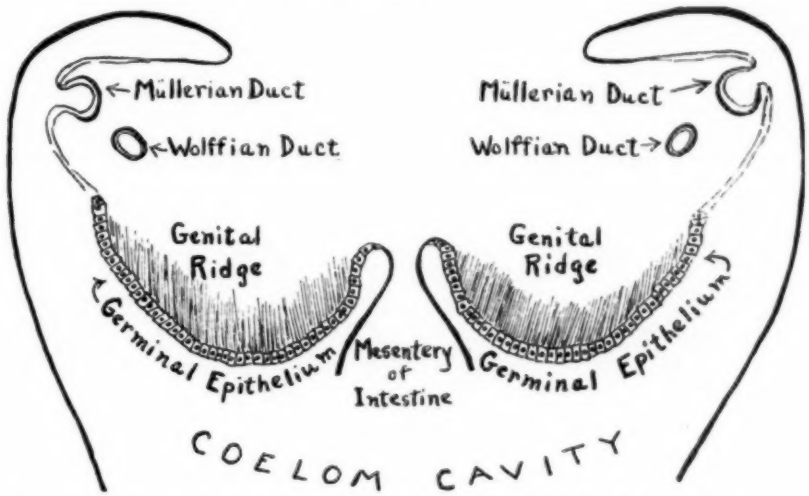

Fig. 120. Schematic representation of the embryological origin of the ovary, Wolffian and Mullerian ducts.

The ovaries take origin from two anlages, the genital ridges, which project into the celom cavity and can be clearly seen in an embryo of two to three months. (Fig. 120.) These projections are covered by a modified celomic epithelium, called germinal epithelium. From this epithelium, cord-like structures grow down into the mesenchyme of the anlage (medullary cords). These primary cord-like structures separate from the germinal epithelium and sink downward into the ovarian substance. A second group of cord-like structures are then formed and they also separate from the germinal epithelium and sink into the mesenchymal substance of the anlage (Pflüger's cords). (Fig. 121.) As one studies these cord-like structures in the mesenchyme, one finds that they differentiate themselves into two types of cells; one becoming the follicular

epithelium and the other the öocyte, which is the precursor of the ovum. At birth the young ovary contains thousands of these primary follicles; consisting of an ovum surrounded by a single layer of follicular epithelium.

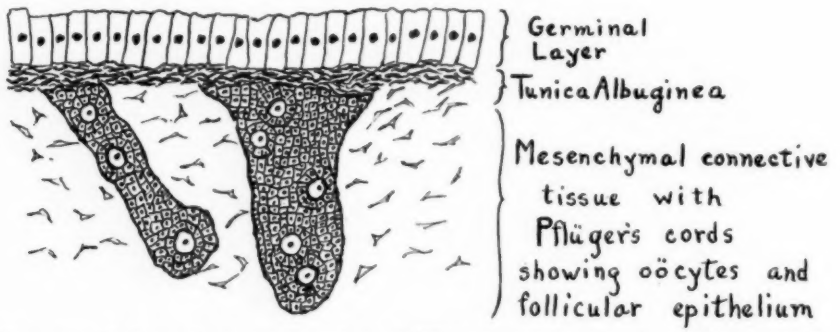
Germinal Layer
Tunica Albuginea
Mesenchymal connective tissue with Pflüger's cords showing oöcytes and follicular epithelium

FIG. 121. Schematic representation of Pflüger's cords, showing follicular cells and öocytes.

To digress for a moment in regard to the origin of these cells: recently there has been considerable agitation as to whether the ova and follicular epithelium develop from the germinal layer of the celomic epithelium. The theory presented is that the ova are derived from the yolk sac entoderm and that the follicular cells are derived from the mesenchymal connective tissue. There is still considerable question as to whether or not sufficient evidence has been produced to prove that the follicular cells are derived from the mesenchymal connective tissue. Unless this is more fully substantiated the original idea will be adhered to; i.e. that the follicular cells and the ova are derived from the celomic cavity epithelium. It is essential, however, to have a knowledge of this new theory, as there are unusual tumors of the ovary which some authors classify according to the theory of the mesenchymal origin of the follicular cells.

The ovary differs from the testis in that at birth it contains all the ova it is every going to have; there is no öogenesis after birth, whereas in the male there is a constant production of spermatozoa throughout the active sex life of the individual. The number of ova found in the ovaries at birth vary in different statistics from 300,000 to 400,000.

Only one ovum is usually discharged with every ovulation. In a woman whose sex life lasts for forty years (thirteen ovulations a year) only 520 ova would reach maturity. The

FIG. 122. Primordial follicle. Ovum surrounded by a single layer of follicular epithelium.

others degenerate and disappear (become atresic). This degeneration or atresia may occur at any stage in the development of a follicle.

The Life History of the Primordial Follicle. The simple primordial follicle consists of an ovum surrounded by a single layer of seven to twelve follicular epithelial cells. (Fig. 122.) When a follicle begins to grow it is mainly the primordial epithelium which proliferates; as a result, an eccentric ovum is seen surrounded by a solid mass of cells. A cavity appears in the mass of follicular cells which is filled with liquor folliculi. This causes a further increase in the size of the follicle. The ovum with its surrounding follicular epithelium forms a mound which projects into this cavity: this projection is called the cumulus oophorus or discus proligerus. (Fig. 123.) The rest of the cavity is surrounded by two or three layers of follicular cells, called the membrana granulosa. In the discus proligerus, immediately surrounding the ovum there is a perfectly regular arrangement of follicular cells, known as the corona radiata. Inside of this is a highly refractile area known as the zona pellucida. Between the zona pellucida and the ovum a small

space may develop called the perivitelline space. During this time the connective tissue just outside the ripening follicle is undergoing definite changes forming a sort of capsule around

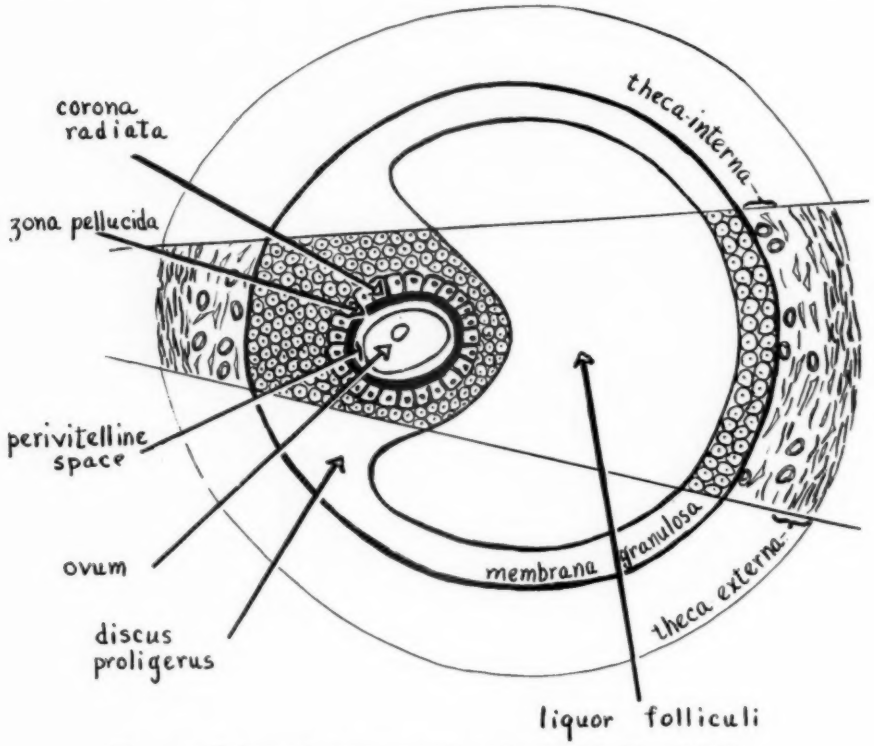

FIG. 123. Schematic representation of a ripe Graffian follicle.

the follicle. This capsule can be divided into two layers, an inner layer, the theca interna or tunical vasculosa, and an outer layer, the theca externa or tunica fibrosa. The inner layer is made up of loose connective tissue containing many capillaries, whereas the outer layer is dense connective tissue which is not sharply demarcated from the surrounding ovarian stroma.

As the Graafian follicle ripens, it sinks into the substance of the ovary and when it is about to rupture, it gradually approaches the surface and rotates until the discus proligerus is just under the point where it will rupture. Occasionally just before rupture, the area of the discus proligerus may be seen floating free in the liquor folliculi. When rupture takes

place, the ovum, zona pellucida, corona radiata and many cells of the discus proligerus are thrown into the peritoneal cavity. Hemorrhage may take place in the area formerly occupied by the Graafian follicle, with the formation of the first corpus; *the corpus hemorrhagicum.* If this does not take place at this time it usually occurs at the next menstrual period.

Shortly after the formation of the corpus hemorrhagicum, large polyhedral cells with central nuclei and granular cytoplasm, arising apparently from the granulosa layer, grow into this area. These polyhedral cells have a yellowish color due to the presence of a lipoid known as lutein and are called follicular lutein cells. This mass of lutein cells form the second corpus; *the corpus luteum.*

At this time, triangular areas of connective tissue are seen growing from the theca interna into this mass of lutein cells. These connective tissue cells are also large polyhedral cells and may contain lipoid; if they do, they are called theca lutein cells. The entire corpus luteum is ultimately replaced by a hyalinized connective tissue with the formation of the third corpus; *the corpus albicans.* The corpus albicans is finally absorbed with resulting scar formation and contraction.

Pathology of the Ovary

Infections. Considering the frequency of pelvic inflammatory disease and the rather small incidence of ovarian infection, one is forced to the conclusion that the ovary is either very resistant to infection or is protected from it by its anatomical position. Perioophoritis with a thickening of the tunica albuginea is not uncommon, but true abscess of the ovary is infrequent. It is not unusual to find a fairly normal ovary with pus tubes or other pelvic inflammatory masses. Abscesses of the ovary do develop: however, one more frequently finds the ovary forming part of the wall of a tubo-ovarian mass. When infection in the ovary proper does occur it takes place either in a Graafian follicle, a cyst, or in a

corpus luteum, thus forming a true ovarian abscess by direct extension. This may occur early in a tubal infection while the fimbriated end of the tube is still patent, or it may be the result of a lymphatic extension from an infected uterus. Direct blood stream infection may occur but it is rather unusual. Microscopically the picture is similar to infection anywhere else.

There is one point of *clinical importance* that should be brought out at this time. When the ovary proper becomes infected or an abscess develops in the ovary as part of a tubo-ovarian mass, the patient's temperature as a rule does not reach normal, under palliative therapy, within a reasonable period of time. In other words, *if the temperature of a patient with a tubal infection does not subside under the usual palliative therapy in from two and one-half to three weeks, one may be reasonably sure that an ovarian abscess is present.*

True Cysts of the Ovary*

A. Follicular cysts;
   1. Simple,
   2. Luteinic.
B. Corpus lutein cysts.

Simple follicular cysts: A true cyst is a degenerative process. In the ovary there is a constant degeneration of unused follicles. Very often circulatory changes in the ovary or a peri-oophoritis with its associated thickening of the tunica albuginea, interfere with the ripening and subsequent degenerative or atresic changes in the follicles. As a result the ova disappear from the follicles and leave a small cavity filled with fluid and lined by follicular epithelium. The small polycystic ovaries, so frequently seen, are made up of several of these atresic cysts. These ovaries usually give very few symptoms and as a rule require no surgery.

Follicular lutein cysts: Occasionally in patients with fibroids, hydatidiform mole or carcinoma of the uterus,

*The basis of this classification was established many years ago by Pfannenstiel and Oscar Frankl.

some of the follicular cells in these simple cysts undergo luteinization, causing part of the cyst wall to appear yellow on gross examination. These cysts are called follicular-lutein cysts. With the removal of the fibroids, hydatidiform mole or the carcinoma, these cysts disappear.

Corpus lutein cysts: If there is some interference with degenerative changes in the corpus luteum, a corpus luteum cyst may develop. They may be recognized by the fact that all the cells surrounding the cavity of the cyst are lutein cells. A corpus luteum cyst may cause an amenorrhea and an inhibition of ovulation with resulting sterility. This may last for a considerable period of time, until the cyst disappears or is destroyed. The secretion of the lutein cells probably acts as an inhibitor to the pituitary and thereby prevents the maturation of the primordial follicles. (See Fig. 97.)

True Tumors of the Ovary

The ovarian tumors will first be classified from the standpoint of their origin, benignity or malignancy. Later their pathology and clinical significance will be discussed.

Origin	Benign	Malignant
Stromatogenous.....	Fibroma Myoma	Sarcoma
Parenchymatogenous.....	Cyst-adenoma 1. Serous 2. Pseudo-mucinous	A. Cyst-adenoma 1. Serous 2. Pseudo-mucinous B. Solid carcinoma (primary) C. Metastatic adeno-carcinoma (Krukenberg tumor)
Embryonic rests.....	Dermoid	Teratoma Dermoid

To this classification are now added five other tumors, but owing to the tremendous amount of discussion as to the exact origin of these tumors they have not been included in the above classification.

1. Brenner tumor.
2. Granulosa cell tumor.
3. Theca cell tumor.
4. Arrhenoblastoma.
5. Dysgerminoma.

What is the pathology and clinical significance of ovarian tumors?

1. *Stromatogenous Tumors.* The stroma of the ovary is made up of connective and some muscle tissue. Benign tumors of these tissues are, therefore, fibromas or myomas and the malignant tumors are sarcomas. Fibromas may be unilateral or bilateral. They may involve almost the entire ovary and may reach a fairly large size. Fibromas as a rule have a moderately long pedicle and as a result of floating in the peritoneal cavity with the accompanying irritation and frequent torsion of the pedicle, ascites may be found.

The malignant tumors taking origin from the stroma cells are the sarcomas. When a sarcoma develops there is usually an early involvement of the mesentery of the ovary with a fixation of the tumor, so that it cannot move about freely.

The clinical diagnosis of these tumors is very difficult. Histologically they can be recognized but many tumors which were previously placed in the sarcoma group now on re-examination and re-classification with our present increased knowledge, are placed in the group of the five unclassified tumors.

2. *Parenchymatogenous Tumors.* The parenchyma of the ovary is made up of primordial follicles. If these cells proliferate, an adenoma is formed. If fluid collects in these epithelial-lined spaces, it is called a cyst-adenoma. If the epithelium grows rapidly and is thrown into papillary formation, it is called a papillary-cyst-adenoma. The fluid in the cyst-adenomas varies. It may be serous or pseudo-mucinous. The epithelial lining of these cyst cavities also varies according to the fluid content. The serous cyst is lined by cuboidal epithelium, and the pseudo-mucinous cyst is lined by high columnar epithelium.

Regarding the origin of the cyst adenomas: there are two schools. One school contends that they take origin from the embryonal rests in the ovary. The other group believe that they take origin from the follicular epithelium and they have attempted to show actual transition from the follicular epithelium of the ovary to the pseudo-mucinous epithelium of the cyst-adenoma. The embryonal rest theory seems to be the more generally accepted view.

Practically all malignant ovarian tumors possess certain peculiarities and these are particularly exemplified by the cyst adenomas. The cyst adenomas are classified as both benign and malignant. *They very rarely metastasize through the lymphatics.* Metastasis when they do occur, are by direct extension. As a rule in ovarian malignancies, if the entire tumor mass can be removed without rupture, they will not recur. Microscopically the tumor may appear most malignant and yet if the capsule is intact, recurrences do not occur. When papillary growths rupture through the capsule of the mass or the ovarian tumor ruptures, epithelial metastasis are found studding the peritoneum.

The generally accepted treatment for malignant ovarian tumors is to remove as much of the ovaries and tumor masses as possible, even if generalized peritoneal metastasis are present. Following this the patient should be treated with *x*-ray and radium. These peritoneal metastasis may disappear spontaneously following the removal of the ovaries.

The second type of malignant parenchymatogenous tumor is the solid carcinoma or solid cancer of the ovary. It apparently takes origin from the epithelial elements of the ovary and grows like carcinoma anywhere else in the body. It is an infrequent tumor and is usually not diagnosed, except in the pathological laboratory.

The third type: metastatic adenocarcinoma. This tumor is usually of the mucinous type. It is frequently classified under the heading of Krukenberg tumor. (Fibrosarcoma mucocellular carcinomatodes.) It is a metastatic tumor, the primary growth occurring somewhere in the G.I. tract. The primary growth may be so small as not to be recognizable. These tumors are

usually bilateral. The ovaries are enlarged and tend to retain their original shape. They look milky-white and have a perfectly smooth surface. They may or may not have cysts in their walls. Whenever one finds a bilateral smooth tumor mass where the ovaries should be, always consider the question of a metastatic ovarian tumor and look for the primary growth somewhere in the G.I. tract. Ascites may be present.

3. *Embryomata.* 1. Teratomas.
2. Dermoids.

Accurately speaking all dermoids belong to the teratoma group of tumors. They are differentiated, however, because of their clinical differences:

Dermoid	Teratoma
1. Develop early in sex life frequently before sexual activity has started.	1. Develop late in sex life usually after sex activity has ceased.
2. Cystic tumors.	2. Solid tumors.
3. Usually contains tissue in a mature state.	3. Usually contains the embryonal tissue of more than one layer (mesoderm-entoderm) in an immature state.
4. Definite tissue formation hair, teeth, bone, thyroid.	4. No tissue formation; usually a mass of cells.
5. Usually benign, rarely malignant.	5. Usually malignant, rarely benign.

The five unclassified tumors:

1. *Brenner Tumor.* "Fibro-epithelioma mucinosum benignum ovarii"—Plaut.

This is a type of ovarian tumor, rather rare, and practically never diagnosed except in the laboratory. It is a benign tumor of the ovary in which the fibrous tissue predominates the picture. In this fibrous tissue there are numerous nests of clear epithelial cells with occasional cavity formation in the cell masses. These tumors do not recur after removal and cause no apparent change in the endocrinological picture in the patient.

2. *Granulosa cell tumor.*

3. *Theca cell tumor.* (Late sex life.)

These tumors of the ovary must be grouped together clinically because of their similar biologic properties. Although histologically they can be distinguished, clinically they resemble each other very much.

This group of tumors apparently take origin from the granulosa and theca cells. They may be found at any time in the sex life of the individual. In the granulosa cell tumors, a small group (10 per cent) occurs before the sex life of the individual has been established. Cases of granulosa cell tumors of the ovary have been reported as early as the second, third and fourth years of life. About 42 per cent occur during active sex life and about 48 per cent occur after the menopause. The theca cell tumors occur more often after sex life has been established or after the menopause.

Microscopically the granulosa cell tumors may vary very much, from the undifferentiated cell of the embryonal epithelium of the young ovary to the fully differentiated granulosa cell of the ripe Graafian follicle. The theca cell tumors are made up of connective tissue bundles. The histological picture of these tumors may be that of a very malignant neoplasm, yet on removal they usually do not recur. Cases have been reported of recurrences (local) being removed, followed by a cure.

Both of these tumors produce large quantities of female sex hormone (theelin) and the peculiar clinical picture presented by these tumors is due to the presence of a superabundance of this hormone.

Clinical features: When these tumors develop before the normal onset of sex life, they produce a precocious puberty. In other words, a child of 3 or 4 will develop all the characteristics of adult sex life—hair distribution, breast enlargement, and menstruation. If these tumors occur in patients after the menopause, there is a reawakening of sex life and a return of menstruation. Removal of these tumors causes a regression of these sex characters; a recurrence of the tumor causes a recrudescence of the symptoms.

4. *Arrhenoblastoma.* These tumors are supposed to take origin from the rete ovarii, the testicular remnants in the ovary. Histologically, they have the characteristics of the testicular elements. They are found in true and pseudohermaphrodites and may be found in apparently normal

individuals. They are supposed to produce a certain amount of internal testicular secretion, thus causing a masculinization of the female with the production of the secondary male characteristics: male voice, carriage, hair distribution and loss of breasts. Following the removal of these tumors, the male characteristics disappear.

5. *Dysgerminoma.* This group of tumors are presumed to develop from the undifferentiated celomic epithelium and histologically may take on the characteristics of either a carcinoma or a sarcoma. They do not produce any female sex hormone (theelin). They are of low grade malignancy and occur at any age in the sex life of the individual.

It is, of course, understood that all of these tumors present pelvic masses, pressure symptoms, ascites, etc. The differentiation between the various types and the proper classification may require the services of several expert pathologists.

Question: What are the conditions which cause a return of bleeding, per vaginam, after the menopause?

Answer: 1. Carcinoma of the uterus, cervix, vagina.
2. Cervical or uterine polyp.
3. Senile vaginitis.
4. Ovarian tumors.

Always examine the vagina and cervix with a speculum in all cases, especially those of post-menopausal bleeding.

Question: Where does struma ovarii fit into this conception of ovarian tumors?

Answer: Struma ovarii is regarded as the development of a tissue in a dermoid or teratoma, just the same as hair, teeth, skin, etc.

Chapter X

ENDOMETRIOSIS

Endometriosis is a neoplasm or tumor growth of aberrant or misplaced endometrial tissue which is peculiar to the female and has never been found in the male. *Histologically*, it consists of a tissue which resembles the epithelium of the endometrium and its underlying stroma. *Physiologically*, with regard to menstruation, pregnancy and the menopause, it apparently is governed by the same laws in its reaction to the glands of internal secretion as is the endometrium lining the uterine cavity.

What is the etiology of these endometrial proliferations?

There are five theories with regard to their origin. No single theory can explain the various forms of heterotopic endometrial tissue.

1. *The Invasion Theory, the "Adenomyoma of Cullen."* Cullen has shown that there is a direct invasion of the uterine muscular wall and other tissues by the uterine glands. Histologically he was able to demonstrate a connection between these glands in the uterine wall and the endometrium.

2. *Implantation Theory.* This theory is most popular and was advanced by Sampson. He believes that there is a retrograde migration of the endometrium from the uterine cavity through the tubes, with an implantation of this endometrium on the various structures in the peritoneal cavity. Several objections have been raised with regard to this theory. It has been held that (1) the endometrium of menstruation is not a viable tissue and therefore could not grow; (2) that it is bathed by menstrual blood which contains trypsin, a proteolytic enzyme, which should prevent its growth, and (3) that the transmural lumen of the tube (i.e. that portion which goes through the uterine wall) is so small that it would be practically impossible to consider some of the large pieces of endometrium found in the tube as coming from the uterine cavity. Novak believes that these pieces of endometrium could

only have been sucked into the lumen of the tube from the peritoneal cavity.

Opposed to this are the experiments of Jacobson, who

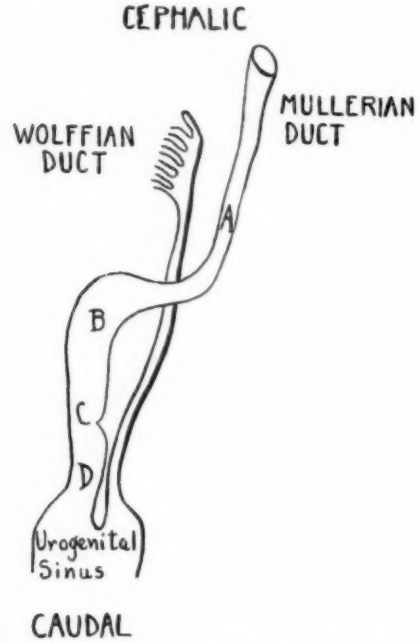

FIG. 124. Schematic drawing, showing relationship of Wolffian and Mullerian ducts. The cephalic portion of the Wolffian duct later forms Gartner's duct. The various parts of the Mullerian duct go to form A, the Fallopian tube; B, half of the uterus; C, half of the cervix; D, half of the upper two-thirds of the vagina.

implanted endometrium from the uterine cavity of a monkey into the peritoneal cavity of the same animal and had it grow; and of Gay, who cultured menstrual endometrium discharged from the uterine cavity during menstruation, in vitro, and it grew.

3. *Direct Transplantation Theory.* This explains the cases where the endometrium is found in scars in the abdominal wall. Two such cases of endometriosis of an abdominal scar have been personally observed following a one-point fixation of the uterus to the abdominal wall. At the point where the

needle went through the abdominal wall, a mass of endometrial tissue grew.

4. *Developmentally Displaced Mullerian or Wolffian Duct Tissue.* This was probably the first theory advanced and was presented by von Recklinghausen. Embryologically, the Mullerian duct at its cephalic extremity develops to the outer side of the Wolffian duct; it then crosses that duct, lying in front and on top of it and finally ending on the inner side of it. (Fig. 124.) Von Recklinghausen's theory was, that, where the Mullerian and Wolffian ducts cross, some remnants of one or the other ducts (embryonal rests) remain and later develop into glandular tissue.

5. *Metaplasia of the Peritoneum.* Ivanhoff, Meyer, Novak and Lauber have advocated this theory, stating that the endometrial implants are the result of a metaplasia of the serosa of the peritoneum, possibly the end result of an inflammation or of ovarian hormonal stimulation. A review of the embryology of the ovary and the Mullerian ducts shows that they are both developed from a modification of the celomic epithelium. (See Fig. 120.) Consequently, the endometrium, the endosalpinx and the follicular epithelium of the ovary are simply degrees of differentiation of the celomic epithelium. Should some undifferentiated or primitive celomic epithelial rests remain anywhere in the pelvis, one might, as Novak states, "get an awakening of the genetic possibilities in some of the unused celomic rests, when stimulated at the proper time by specific forces." Inflammation or ovarian secretion could very well be the stimulating factor.

Where are these endometrial implants found?

Cullen has shown the various areas where endometriosis is found: (1) adenomyoma of body of uterus; (2) adenomyoma of rectovaginal septum; (3) adenomyoma of uterine horn or Fallopian tube; (4) adenomyoma of round ligament; (5) in hilum of ovary without a myomatous growth; (6) in utero-ovarian ligament; (7) in uterosacral ligament; (8) in sigmoid flexure; (9) in rectus muscle and (10) adenomyoma of the umbilicus.

How often does endometriosis occur?

In women who have non-infected pelvic organs, the frequency of endometriosis is in direct proportion to the care with which the examination is made. The finding of these implants does not necessarily mean that they are giving symptoms.

What is the gross pathology of endometriosis and how can it be recognized?

They appear early as small blue nodules, no larger than the head of a pin, on the uterus, ovary, rectovaginal septum, etc., with dense, puckering adhesions around the small blue nodule. Endometriosis is rarely single; it is always multiple. These endometrial areas react to the ovarian hormones in the same manner as the uterine endometrium, so that with each menstrual period bleeding takes place in these areas. This blood is very irritating and causes dense adhesions. The denseness of the adhesions formed by endometriosis is equalled only by carcinoma. This is evidenced by the fact that until the gross pathology of this lesion began to be recognized and described by such men as Cullen, Sampson and others, the large intestine was frequently resected for suspected carcinoma. When the lesion occurs in the rectovaginal septum, the blue dome cysts may occasionally be seen in the vault of the vagina with a speculum.

With every period there are fresh exacerbations and implants. Later these implants rupture and disseminate over the pelvic peritoneum, sigmoid, uterosacral ligaments and even anteriorly over the bladder.

Occasionally the blood is walled off by adhesions, thus forming cysts with fairly thick walls and dense adhesions—except at one spot where the cyst wall is usually very thin, so that on attempting to remove the cyst, it ruptures and a thick, dark brown, tarry material is extruded (chocolate cyst).

If the operative specimen is put in formalin before examination, the endometriosis areas lose their blue color and cannot be recognized. If one wishes to make a diagnosis from gross pathological material, fresh tissue must always be examined.

What are the symptoms of endometriosis?

Endometriosis has never been found before the development of active sex life. The symptoms are not from the endometrial implants themselves, but are attributable to the body's reaction to the irritating blood extruded by these endometrial implants with every menstrual period. The most frequent symptom is a gradually increasing dysmenorrhea of such severity that a patient stated; "Doctor, I don't care what you find, you must remove everything you find that is pathological; the pain is getting more severe with each period, with the result that now something must be done."

The symptoms are periodic and progressive; they occur at the beginning and just after the menstrual period. In the two cases of endometriosis (transplantation) in an abdominal wall scar, the patients stated that the tumor mass grew larger at each menstrual period; following which it receded, but never to the point where it was before. Increasing constipation, worse at the time of menstruation, is a not infrequent symptom. Other signs and symptoms such as tumor, cystic ovaries and a firm, resistant, nodular cul-de-sac are due to associated lesions, adhesions and collections of blood. A sudden, acute abdominal catastrophe may occur as a result of a rupture of one of these collections of blood.

How can one make a diagnosis of this condition preoperatively?

Axiom: In order to make a diagnosis of any condition one must first be aware that it occurs.

When a patient presents herself with a gradually increasing acquired type of dysmenorrhea, occurring mostly after the menstrual period, think immediately of endometriosis. Given a patient in whom one can exclude pelvic inflammatory disease, who presents an increasing acquired dysmenorrhea, indurations in the cul-de-sac with or without an adherent, fixed retroflexion, constipation worse at the time of menstruation, the diagnosis of endometriosis must be seriously considered. Rarely one may see the blue domed cysts in the vault of the

vagina. This, when associated with the typical symptomatology, is pathognomonic.

When the diagnosis is made, or if on opening the peritoneal cavity for some other condition, endometriosis is found, what treatment shall be instituted?

The treatment depends upon three factors: (1) The age of the patient; (2) the desire on the part of the woman to retain her capacity for becoming pregnant; and (3) the type of lesion found.

CASE I. A young woman with endometriosis, marked adhesions between the rectovaginal septum and the posterior wall of the uterus, severe dysmenorrhea and anxious to become pregnant.

The patient is placed in high Trendelenburg position, a left paramedian incision is made and the intestines walled off. With good light and exposure, the adhesions between the uterus and the rectosigmoid are separated by sharp dissection. *If necessary, leave a part of the uterus on the rectosigmoid rather than a piece of the rectosigmoid on the uterus.* The separation of these adhesions releases the uterus, which must be held well out of the pelvis. For this purpose an Olshausen suspension seems to work well. The ovaries, which are usually involved, are also freed and suspended by non-absorbable suture material to the parietal peritoneum above the iliopectineal lines (Poole suspension). Occasionally, a large raw area may be left in the cul-de-sac.

On discharge the patient is advised to become pregnant as soon as she can, as one cannot tell when her symptoms may return and castration becomes necessary. Peculiarly, these patients have great difficulty in becoming pregnant. There seems to be something which lowers their fertility, but just what causes it is not known.

CASE II. A woman in the childbearing period who has endometriosis and has had children, but does not wish to have her ovaries removed.

That patient can have the same operative procedure as Case I with the understanding that, should her symptoms return, she will promptly receive a castration dose of *x*-ray, which would stop the function of her ovaries and prevent further dissemination of the endometriosis. Knowing the diagnosis, one may always take a chance and leave the uterus and ovaries in, because on the recurrence of symptoms one can always have recourse to a castration dose of *x*-ray.

CASE III. A patient late in her sex life, in the late thirties or early forties, with an endometriosis.

This patient should have both ovaries removed and as much endometrial tissue as possible, without damaging any of her other organs. This

will stop any further pathology, prevent recurrence of the symptoms, and will cause those endometrial implants present to involute.

Question: What effect has a simple hysterectomy, leaving both ovaries?

Answer: None, as the implants depend for their continuance of function on the ovaries. Removing the uterus alone would not cure the condition.

Question: Should a hysterectomy be performed on these patients?

Answer: No. Removing the uterus does not cure the condition. To cure the condition, one must stop the ovaries from functioning. The loss of the internal secretion of the ovaries stops the growth of the endometrial implants.

Question: In a young woman, is *x*-ray castration sufficient?

Answer: 1. Yes, an *x*-ray castration dose will stop any further development of the pathology, but it will not cause the pathology that is present to disappear.

2. It is difficult to castrate a young woman with *x*-ray. Amenorrhea may be produced for a period of time, but at the end of that time the patient will again menstruate. Should the symptoms recur, a second castration dose can be given.

Question: Does *x*-ray have any effect on the masses themselves?

Answer: No, because the mass is composed of adhesions and blood.

Question: Does endometrial tissue occur in the tubes?

Answer: Yes, Novak has definitely shown areas of endometrial-like epithelium lining the lumen of the tube.

Question: Is curettage of any value in this condition?

Answer: No.

Question: Do you often get tarry stools in such cases?

Answer: No, unless there is a rupture of a chocolate cyst into the rectum.

Question: Can endometriosis be prevented?

Answer: Curtis goes so far as to say that every patient with a third degree retroflexion should be operated upon and have a suspension performed to prevent endometriosis. He

believes that a third degree retroflexion, according to Sampson's theory of retrograde spilling of the blood, is the etiological factor of endometriosis.

Question: Do all cases of endometriosis require surgical or *x*-ray castration?

Answer: No, the majority of cases of endometriosis found are small, asymptomatic and of academic interest only. They may later cause more severe symptoms. Therefore, when the condition is accidently seen at operation, it should be recorded in the operative findings.

Question: Are all blue domed cysts endometrial cysts?

Answer: No, there are other blue domed cysts due to clear fluid. The blue domed endometrial cyst, when punctured, is found to be filled with blood or tarry fluid.

Chapter XI

CARCINOMA

Practitioners are interested in carcinoma from three angles:

First: The diagnosis. Is the lesion which the patient presents malignant?

Second: If it is malignant, what is the degree of malignancy? Is it a "good" cancer or a "bad" one? Will the patient die as a result of the cancer within six months or will she live for years?

Third: Having already established the diagnosis, and knowing the degree of malignancy, what form of therapy should be advised; surgery, radiation, or both?

"*Has the patient a carcinoma?*"

The patient presents herself with a lesion of the cervix. The cervix will be used as the first example, because it is the most accessible and it is the place in the uterus where carcinoma occurs most frequently. One can read reams of literature on the clinical differentiation of benign from malignant lesions of the cervix, but in the final analysis the diagnosis is established by a biopsy. For this purpose it is advisable to have a punch (of the nose and throat variety) with a basket arrangement at the base to hold the tissue. (Fig. 125.) The piece of tissue removed should be immediately placed in 10 per cent formalin. There are two precautions to be observed when taking a punch biopsy; first, be sure to remove a piece of the diseased tissue; second, when possible have the biopsy include a piece of normal tissue so that the transition from the normal to the pathological may be studied. The tissue is taken and sections made for microscopic study.

How is the diagnosis of carcinoma made from the microscopic section? What criteria are used?

To make a diagnosis of malignancy, three questions must be answered affirmatively:

1. Are the cells atypical?
2. Have the cells an atypical arrangement?
3. Have the cells an atypical relationship to the surrounding structure?

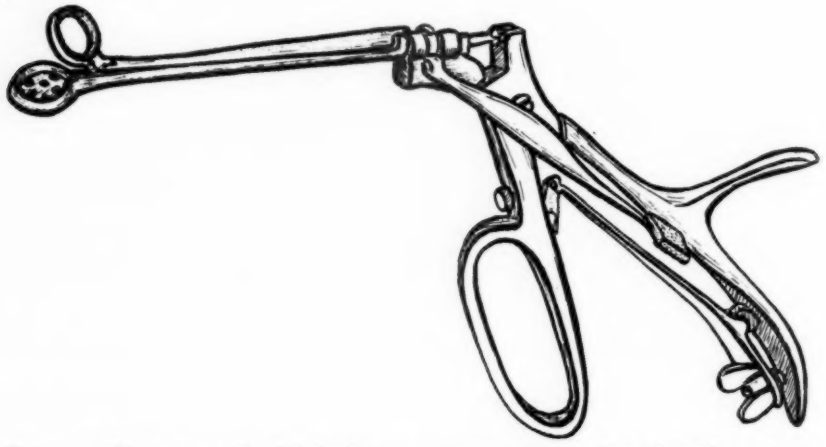

FIG. 125. Biopsy punch with basket arrangement to catch and hold specimen.

What is meant by an atypical cell? To define an atypical cell, one must first give the criteria of a typical cell. Typical cells are those that are found in normal tissue under normal circumstances. Atypical cells are those that are never found in normal tissue under normal circumstances. A tumor which is made up of typical cells is a benign tumor. A tumor which is made up of atypical cells is a malignant tumor.

What is meant by an atypical cell arrangement? In a stratified squamous epithelium the epithelial cells are arranged in layers. (Fig. 126.) The basal layer takes a deep stain with hematoxylin—layer A. It is perfectly regular, usually cuboidal in form with a central nucleus. The next layer, layer B, may or may not be nucleated. The cells are beginning to lose their cuboidal shape. The next layer, layer C, is usually without nuclei; and layer D is the hornified layer on the surface. The cells are arranged according to age—layer A is the youngest and layer D the oldest. When layers A, B, C or D are referred to, one is not speaking of any single cell, but of a group or layer of cells. This is the typical arrangement that is found in stratified squamous epithelium. Should this arrangement be

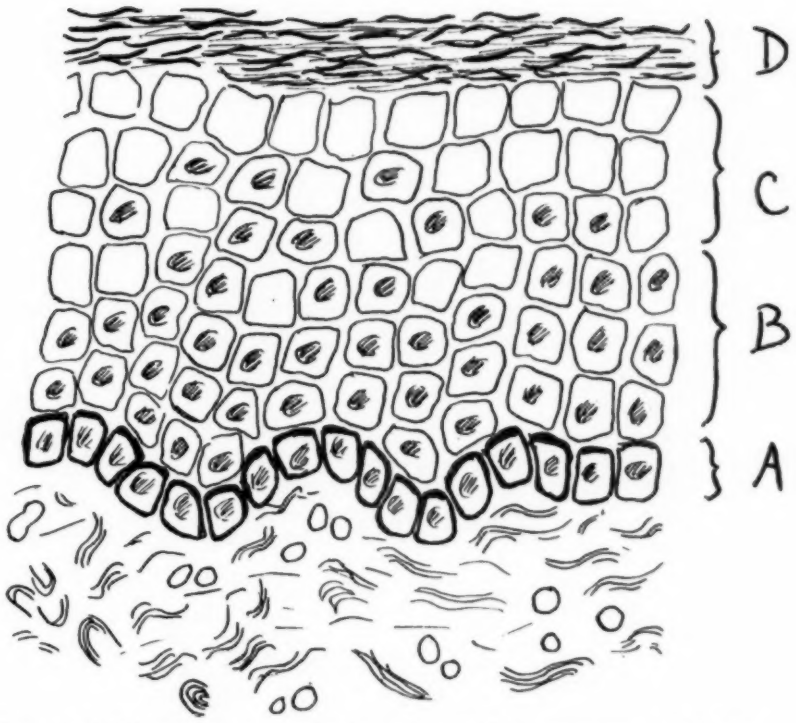

FIG. 126. Schematic representation of stratified squamous epithelium. (Typical arrangement.) A; basal layer. Regular arrangement and deeply straining nuclei, sharply demarcated from the surrounding tissue. B; second layer. Cells are slightly older. C; third layer. Cells are still older and frequently lack nuclei. D; hornified surface layer. No cellular structure.

FIG. 127. Epithelial pearl, showing atypical cell arrangement. Hornified layer surrounded by fairly young epithelium.

disturbed and one were to find cell D (the hornified layer) arranged in whorls with a layer of young epithelial cells around it from group A, this would be an atypical cell arrangement for squamous epithelium. (Fig. 127.)

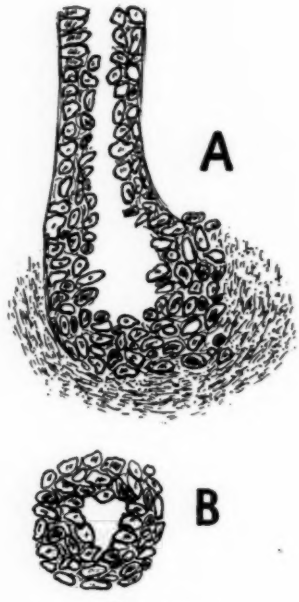

FIG. 128. FIG. 129.

FIG. 128. A. Normal gland, sharply demarcated from the surrounding structures-typical relationship with surrounding tissue. B. Cross section of gland showing single layer of cells. Typical cell arrangement.

FIG. 129. A. Atypical relationship with the surrounding tissue; breaking through and ramification of glandular epithelium into surrounding tissue. B. Cross section of gland showing an atypical cell arrangement; epithelium in several layers.

In a gland, the cells are normally arranged in a single layer lining the cavity of the gland. (Fig. 128.) If instead of finding a single layer of cells, one finds the gland epithelium thrown up into three or four layers, one speaks of this as an atypical arrangement of cells. (Fig. 129.)

When can one say that the cells have an atypical relationship with the surrounding structure? The typical gland and the typical stratified squamous epithelium must again be used. The typical gland is sharply demarcated from the surrounding tissue. (Fig. 128.) If, however, the cells at the fundus

are piled up and ramify into the surrounding structure, this is an atypical relationship of the cells to the surrounding structures. (Fig. 129.) In stratified squamous epithelium, the

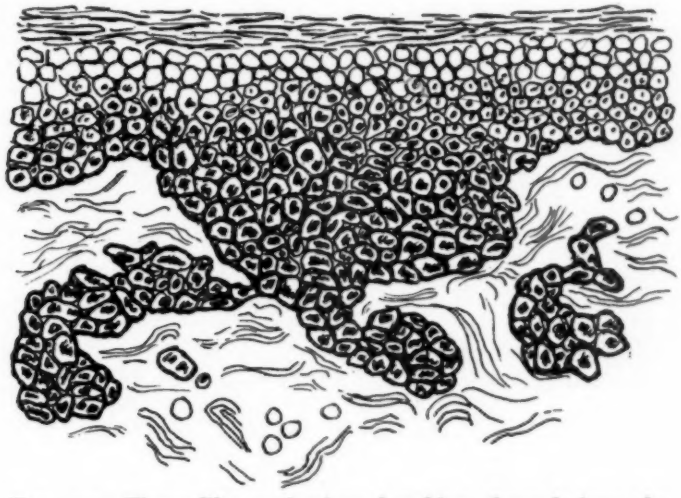

FIG. 130. Finger-like projections breaking through into the surrounding tissue showing atypical relationship with surrounding structures in stratified squamous epithelium.

epithelium in the basal layer undulates and is sharply demarcated from the surrounding structures. (Fig. 126.) If, instead of these undulations and sharp demarcations, one finds finger-like projections breaking through into the surrounding tissue, one speaks of this as an atypical relationship with the surrounding structure. (Fig. 130.) If some epithelial cells are found in a lymph space or a blood vessel, this also is an atypical relationship with the surrounding structure. Epithelial cells found in the connective tissue without any apparent connection to any other epithelial tissue, is also spoken of as an atypical relationship with the surrounding structure.

Having examined the slide very carefully with the three questions in mind, in order to make a diagnosis of carcinoma, *all three questions must be answered in the affirmative.*

Can a diagnosis of carcinoma be made if only two questions are answered in the affirmative? No.

Patients are frequently seen with lesions which are the result of inflammatory reaction, healing erosion or eversion

where the reparative process causes the cells to have an atypical arrangement. This process may even go so far as to present an apparent down growth of epithelium into the connective tissue giving the appearance of an atypical cell arrangement. These are the end-results of an old infection or a healing lesion. There is no question that inflammation may cause lesions to develop that so closely simulate carcinoma histologically as to make the diagnosis exceedingly difficult even for expert pathologists. They may therefore request several biopsies from different portions of the tumor before they are able to give a positive diagnosis. The diagnosis of carcinoma, however, should not be made unless one can answer all three questions in the affirmative.

If my patient has a carcinoma, is it a "good or a bad one?" What is the degree of malignancy and how can it be determined?

In spite of all the research work on carcinoma, only one fact with regard to its etiology is known and that is that *wherever there is chronic irritation, carcinoma is more apt to develop.* It has been definitely shown that chronic irritation, even at a point where the occurrence of carcinoma is unusual, may cause the development of a tumor in an individual who is susceptible to carcinoma. This does not mean that wherever there is chronic irritation a carcinoma must develop; but it does prove that there is a definite relationship between chronic irritation and carcinoma.

Why should a patient be more apt to develop a carcinoma on an area of chronic irritation? This discussion is purely theoretical and for this purpose stratified squamous epithelium will again be used as an example. As a result of the chronic irritation there is a loss of the superficial hornified layer D. (Fig. 131D.) When this takes place, cell C moves up to take the place of the hornified layer D. Cell B moves up to take the place of cell C, and cell A moves up to take the place of cell B. When this happens a stimulus goes to some unknown point called "X" that a new cell is needed. The new cell which is produced to take the place of A will be called A′. As a result of

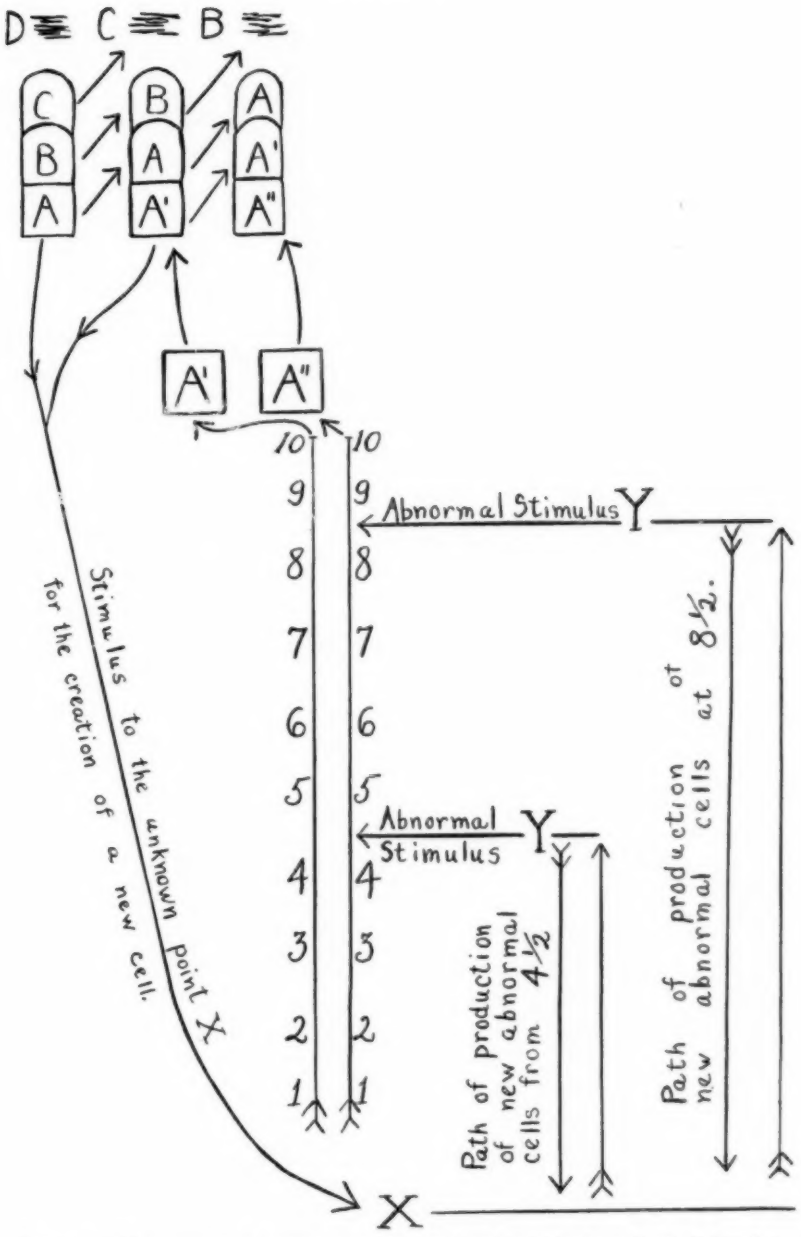

FIG. 131. Diagrammatic representation of the theoretical method of development of cancer cells. A schematic presentation showing how normal cells, A'-A" replace normal cells, and how an abnormal stimulus, Y-Y produces carcinoma cells at various points in the cell development.

further irritation, the new layer C is destroyed; B moves up to take the place of C; A to B; A′ to A and a stimulus is again sent to point "X" for another cell. As the irritation continues, these new cells are constantly being formed. When the stimulus is sent from A, A′, etc. to "X" to form a new cell, the new cell does not suddenly spring out fully formed. This new cell must of necessity go through certain phases in its development, before it can become the adult cell A, A′ or A″. (Fig. 131.) For discussion purposes, it is assumed that the cell goes through ten phases. This is an arbitrary, theoretical division in order to simplify the discussion. The stimulus received at "X" is normal, and the cell being produced at "10" when it goes through the ten phases will be a normal cell. But suppose that somewhere during the process of the development of the new cell, an abnormal stimulus "Y" is received. (Fig. 131.) This stimulus may be enzymal, thermal, bactericidal, or chemical —just what this stimulus is is not known, but its effect is known. If this abnormal stimulus "Y" is received at point "4½," it causes the cell to stop developing beyond that point, and to begin to reproduce in kind.

The cells thus produced are abnormal pathological cells. In other words, the abnormal stimulus causes the production of an atypical, immature cell, i.e. a *carcinoma cell.* If this abnormal stimulus was received at point "4½" in the development of the cell, it would produce a pathological cell low in the scale of development. The new cell which is being formed has not gone through half of its normal developmental phases. When this immature pathological cell reproduces, it cannot produce a cell beyond the "4½" point of development, so that the new cells produced from this nidus are cells of low maturity. Tumors which are made up of cells of low differentiation are called *carcinomas of low maturity.* Carcinomas of low maturity are histologically relatively malignant tumors. These tumors grow rapidly because it takes a comparatively short time for the cells to reach their peak of differentiation at "4½" and to reproduce.

What are the histologic signs by which these cells can be recognized? They can be recognized by their resemblance to

the embryonic cell. In the low maturity cell, the nucleus is very irregular in size; it may be small, pyknotic, or large, megalocytic. The nucleus may be single, multiple or mitotic. The more rapidly growing the tissue, the more mitotic figures are seen. The cytoplasm of these cells varies in amount from a very small quantity, barely enough to surround the nucleus, to a large amount. The cell outlines are irregular and indefinite. There is a marked hyperchromatism of the nucleus, these young cells taking large amounts of hematoxylin. The nuclei may be irregularly stained and may show striations.

If the abnormal stimulus "Y" is received by the developing cell at point "8" in its development, an abnormal cell would be produced which would so closely resemble the fully formed cell at point "10" that it would be almost impossible to differentiate them. These cells of high differentiation reproduce slowly and the new cells formed are also cells of high maturity. Tumors of this type are called *carcinomas of high maturity*. These tumors owing to their slower growth are classified histologically as being relatively less malignant. Occasionally in tumors of high maturity the malignant cells so closely resemble the normal tissue as to make the histological differential diagnosis very difficult; because question 1, "Are the cells atypical?" cannot be answered affirmatively until an area of lower differentiation is found. This may require a long search. There is, of course, a group of tumors between these two, to which the term *middle maturity carcinomas* has been applied.

Carcinomas are made up of cells of various types of differentiation. The histological maturity of a tumor is judged by the predominant type of cell found. Thus the second question is answered. The higher the histological maturity of the carcinoma, the relatively lower is its malignancy; the lower its histological maturity, the relatively higher is its malignancy.

What form of therapy should be advised?

The diagnosis of carcinoma having been made, its histological maturity rating having been established, what advice shall the patient receive as to the treatment for her carcinoma. Here arises a paradox. Carcinomas of relatively high malig-

nancy (low maturity) are more susceptible to *x*-ray and radium than are the low malignancy (high maturity) tumors. Why should a carcinoma which is histologically very malignant be more susceptible to *x*-ray than a low malignancy tumor? An examination of Fig. 131 will help very materially. The difference in the scale of development of a cell at point "10" and one at point "4½" is so great that it gives a wide zone of safety for the lethal dose of *x*-ray and radium; e.g. a dosage of radium or *x*-ray that would be lethal for a cell at "4½" in its development would not kill the cells at point "10." Whereas the difference in development of a cell at "8" and another at "10" is so close, that the lethal dose for a cell at "8" would also be lethal for the cell at "10." Therefore the higher the maturity, the relatively more difficult it is to treat that carcinoma with *x*-ray and radium.

Radical surgery in the form of Wertheim operations, etc., is losing more and more ground daily. The general consensus of opinion in most clinics seems to favor *x*-ray and radium for carcinoma of the cervix; *x*-ray and radium plus total hysterectomy in carcinoma of the fundus.

Clinical Classifications. Carcinoma of the uterus occurs in one of three places:

1. The portio vaginalis.
2. The endocervix.
3. The body of the uterus.

Which of these tumors is the most malignant from the anatomical stand point. This is an anatomical clinical classification and has nothing to do with the histological picture; i.e. the maturity of the cancer cell. Endocervical carcinoma is the most malignant; the next in malignancy is that of the portio vaginalis and the least malignant of the three is carcinoma of the body of the uterus.

Why is the endocervical carcinoma clinically the most malignant?

1. A carcinoma developing in the cervix grows along the path of least resistance. As the cervix does not dilate readily it grows directly into the musculature of the cervix.

2. The lymphatics of the cervix are very profuse, and owing to the rapid involvement of the musculature the lymphatics become involved very early.

3. The glandular tissue of the cervix is thin and rests directly on the musculature. There is no submucosa, therefore, the musculature of the cervix becomes involved very early.

Carcinoma of the body of the uterus is the least malignant of the three types because:

1. The body of the uterus will distend readily to accommodate itself to its contents. Therefore the carcinoma grows toward the cavity of the uterus and thus there is a late involvement of the musculature.

2. The lymphatics of the body of the uterus are few as compared with the cervix and are rarely invaded.

3. The endometrium is thick and allows for a considerable growth before there is any involvement of the musculature.

What are the methods of metastases of carcinoma of the uterus? There are three:

1. Through the lymphatics.

2. By direct extension.

3. By the snaring of portions of the carcinoma by connective tissue. This may be classified as part of Group 2. Inflammatory reaction on the part of the tissues is frequent with carcinoma. This causes a marked increase in connective tissue around the area; and occasionally there is a snaring off of small areas of carcinomatous tissue by the connective tissue, carrying these cells away from the primary growth. These areas then grow independently.

In a study of a large number of carcinomas of the cervix it was found that there was no lymphatic involvement in 45 per cent of the cases. (Schauta.) The entire carcinoma was limited to the local area and had extended only by direct invasion of the surrounding tissue. On vaginal examination, patients with carcinoma of the cervix frequently show large masses in both fornices. These indurated areas fixing the cervix do not necessarily mean metastases—they may be the inflammatory reaction on the part of the tissues to the car-

cinoma and the infection present. When the infection in the carcinoma has been cleared up, a large part of these inflammatory masses disappear.

"Clinically, according to the League of Nations classification, cancer of the cervix can be grouped into four classes representing different stages of the disease.

"Stage I. The growth is limited entirely to the cervix. The uterus is freely movable and no paracervical infiltration can be detected.

"Stage II. The parametrium is involved. The infiltration can be palpated under the vaginal wall or beside the cervix. The palpable infiltration may extend outward nearly to the pelvic wall, but the uterus still retains considerable mobility. This stage includes the old 'border-line operable' group.

"Stage III. The parametrium is involved to the pelvic wall on one or both sides, with partial or complete fixation of the uterus.

"Stage IV. The cancer has invaded adjacent viscera or extensively involved the vagina or produced distant metastases.

"In the case of each patient, the evidence of the extent of involvement should be worked out sufficiently to permit accurate assignment to class. Accuracy is enhanced by deciding definitely as to the class at the time of examination and then recording the decision. This obviates the difficulty encountered when trying later to classify a borderline case with some needed detail missing. This accurate classification is best made during examination under the analgesia for the radium implantation. Deep rectal palpation of the broad ligament of each side is most helpful in determining the fact and the extent of parametrial involvement."*

Symptomatology of Carcinoma

When the usual group of textbook symptoms of carcinoma of the uterus appear (such as irregular bleeding, foul, watery discharge, etc.) the lesion is far advanced. In order to make an

* Crossen, H. S. *Am. J. Obst and Gyn.*, 28: 378, 1934.

early diagnosis—and those are the ones that will be of definite benefit to the patient—it is necessary to make it at a time when there may be few or no symptoms. To do this *every patient presenting herself with a vaginal discharge or slightly irregular bleeding (bleeding or show on intercourse), should have her cervix examined with care and if there is any doubt, a biopsy should be performed.*

Two of the earliest cases of carcinoma that have been observed at the hospital were found as a result of the regular inspection of the cervix of every gynecological patient.

Axiom: The cervix of a menstruating woman need not be examined but one must always look at the cervix of a woman who is bleeding.

Clinically, a carcinoma of the cervix presents itself in one of three ways:

1. As a tumor.
2. As an ulcer.
3. As a combination of both.

Bleeding after the menopause is due to one of four causes:

1. Carcinoma of cervix or uterus.
2. Senile vaginitis.
3. Cervical or uterine polyp.
4. Ovarian (granulosa cell) malignancy.

Question: What about the Schiller iodine test?

Answer: When the normal vaginal epithelium is painted with Grams iodine solution (iodine 1, potassium iodide 2, water 300), it assumes a dark brown color due to the presence of glycogen in the epithelium and its reaction with the iodine. Abnormal epithelium, cancer epithelium, lacks glycogen and therefore does not become dark brown. This test has not been of as much help as was originally anticipated, as many areas which are not carcinomatous, but are healed areas of inflammation or hornified areas, do not take the stain.

Question: Of what value is the colposcope?

Answer: This instrument which throws a bright light into the vagina and allows one to examine the cervix under a × 10 magnification, is of value in finding early denuded areas.

However, the main benefit of both the colposcope and the Schiller iodine test lies in the fact that it makes the practitioner examine the cervices more carefully.

Question: What about pre-cancerous lesions of the cervix?

Answer: Many men describe all forms of cervical lacerations, erosions, eversions and chronic inflammations as pre-cancerous. If they mean that all of these lesions are potential cancers (i.e. can become malignant under the proper abnormal stimulus this can be subscribed to); but if they mean something occurring before every cancer, this cannot be accepted as proven.

Question: Is there any relationship between the maturity of the carcinoma and the age of the patient?

Answer: As a rule there is no connection. However, tumors occurring in very young individuals are more apt to be of lower maturity.

Question: What is the lymphatic drainage of the uterus and vagina?

Answer: The cervix and upper two-thirds of the vagina are drained by one group of lymphatics; the body of the uterus is drained by a separate group as is the lower third of the vagina. The lymphatics of the cervix and upper two-thirds of the vagina go outward into the broad ligament, following the uterine artery to the hypogastric artery, then to the common iliac and the bifurcation of the aorta, and finally along the aorta. There is one lymph node which may be occasionally found in the broad ligament. The second group of nodes is found at the junction of the common iliacs, and the third group is found at the origin of the mesenteric vessels.

The lymphatic vessels of the body of the uterus pass outward through the mesosalpinx to the infundibulopelvic ligament and ascend with the ovarian vessels and drain into the lymph nodes along the aorta.

The lower third of the vagina and the external genitalia are drained by a large number of lymph vessels which communicate with the superficial and deep inguinal glands.

Question: Does an enlarged lymph node usually mean carcinomatous involvement?

Answer: No. Enlarged lymph nodes do not of necessity mean carcinoma. They may be enlarged from chronic infection. The absence of carcinoma from one group of nodes does not necessarily mean an absence of carcinoma beyond these nodes. Carcinoma has been known to jump a series of nodes.

[*To be continued in subsequent issues.*]